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ANTIRACHITIC EFFECT OF COD LIVER OIL FED DURING THE PERIOD OF PREGNANCY OR LACTATION *

ALFRED F. HESS, M.D., AND MILDRED WEINSTOCK

NEW YORK

In view of the fact that cod liver oil has been found to be a specific in the prevention and cure of rickets both in animals and in man, experiments were undertaken to acquire fuller information regarding the scope of its activity. From both a theoretical and a clinical point of view, it seemed of value to ascertain whether, when cod liver oil is fed to pregnant animals, its active principle passes through the placenta and is imparted to the fetus. Furthermore, it seemed worth while to inquire whether the specific factor is secreted into the milk in quantity sufficient to protect the young. From a nutritional standpoint, the period of lactation must be regarded from two different points of view. The one, just referred to, considers solely the antirachitic virtue of the milk; the other takes into consideration supplementary food that the young may receive directly during this period. In this study, these two different aspects were considered by feeding cod liver oil to the lactating mother as well as directly to the young during the period of suckling. These questions were investigated as part of a more extensive study of the influence of diet on the subsequent development of rickets.

The rats used for these experiments were from our laboratory stock and were fed the standard dietary, which consisted of whole wheat, corn, bread and potatoes daily, with additions of oats, chopped beef, greens, milk, banana and cheese; an ample, although not an optimal dietary. The cod liver oil was the refined Norwegian variety which, in the past few years, has been frequently tested out in this laboratory; it titrates 35 drops to the gram. In all tests, whether of mother or of young, the oil was given by pipet and was not incorporated in the food. In the first test, a pregnant rat was given 5 drops of oil for the last twelve days, almost the latter half, of pregnancy. In due time, she had a litter of four. The fact that this litter was so small renders the protective dose of cod liver oil comparatively large. During the period of lactation, the oil was discontinued and merely the standard dietary given.

* Received for publication, Sept. 22, 1923.

* From the Department of Pathology, College of Physicians and Surgeons.

After the usual preliminary interval of four weeks, the young were placed on rickets-producing dietaries. The accompanying table gives the details of this and the other experiments. It will be noted that two rickets-producing diets were fed; the No. 84 (Sherman-Pappenheimer) dietary, and another in which 5 per cent. of the flour was replaced by an equivalent amount of dry milk, dried by the Just-Hatmaker roller process. All four animals of this group developed rickets in the usual degree; in other words, little or no protection had been afforded the young by feeding the pregnant mother cod liver oil.

In the next three tests, cod liver oil was fed to the mother during lactation. In the first test, 5 drops was given for seventeen days, which constitutes more than half the lactating period. As the young did not develop marked rickets, the experiment was repeated, the same amount of oil being given, but fed to the mother throughout lactation, for the entire twenty-eight days. After this interval the young were weaned and placed on one or the other rickets-producing dietary. Four weeks later roentgenograms were taken, and the animals were killed for microscopic examination of the bones. These animals had rickets in a moderate degree, some showing histologic evidence of slight healing, a phenomenon not uncommonly noted in the course of active rickets. In order to obtain more definite information on this subject, a third test was carried out, the mother receiving 20 drops of oil throughout lactation. This amount of oil is exceedingly large, and had to be fed in divided doses in the course of the day. From the point of view of body weight, feeding the mother this amount is equivalent to giving an infant about 180 c.c. daily. As has been stated elsewhere,[1] moderate rickets developed also under these conditions, indicating that the young had derived little benefit from the feeding of cod liver oil to the mother.

In the next test, oil was fed directly to the young. It was given to the little rats by means of a pipet during the latter half (fourteen days) of the suckling period. The litter consisted of eight; one half this number were fed 6 drops of oil daily; the others served as controls. Given directly in this way, cod liver oil exerted a protective action, very slight rickets developing on the standard dietary (No. 84) after the usual test period of twenty-eight days.

It is evident that this result must presuppose a mechanism whereby cod liver oil, or its antirachitic principle, can be stored in the tissues of the young. A phenomenon of this kind is complex, and depends on various factors. Above all, the factor of growth must be taken into consideration in this connection, as in all phenomena relating to the development of rickets. This applies not merely to the active stage of

1. Hess, A. F.; Weinstock, Mildred, and Tolstoi, E.: Proc. Soc. Exper. Biol. & Med. **20**:371, 1923.

Prophylactic Feeding of Cod Liver Oil to Rats During Pregnancy, or to Mother or Young During Lactating Period

Rat	Stock	Oil Fed During Pregnancy	Oil Fed During Lactating Period		Drops of Cod Liver Oil (35 Drops to the Gram)	Weights of Young, Gm.	Rickets-Producing Diet to Young*	Rickets		Remarks
			To Mother	To Young				Roentgenogram (28 Days)	Microscopic (28 Days)	
5205	O.	Last 12 days			5	40-40	84	Moderate	Moderate	4 in litter
5206						38-40		Moderate	Moderate	
5203						34-48	5% dry milk	Slight	Moderate	
5204						38-50		Slight (healing?)	Moderate	
5000	O.		Last 17 days		5	48-50	84	Slight	Slight	8 in litter
5001						44-50		Moderate	Slight	
5002						46-50		Very slight	Slight	
5003						52-44		Slight	Slight	
5004						48-60	5% dry milk	Moderate	Slight	
5005						52-64		Moderate	Slight	
5006						48-58		Moderate	Slight	
5007						48-54		Moderate	Slight	
5252	O		Entire 28 days		5	30-44	84	Marked	Moderate (slight healing?)	10 in litter
5253						34-50		Marked (healing)	Moderate (healing)	
5254						40-50		Moderate	Moderate	
5255						34-56		Moderate	Moderate (slight healing)	
5256						34-40		Slight (healing?)	Moderate (slight healing)	
5257						30-54	5% dry milk	Moderate (marked healing)	Moderate (healing)	
5258						30-54		Moderate (marked healing)	Moderate (healing)	
5259						40-54		Slight	Moderate (healing)	
5260						40-60		Moderate (marked healing)	Moderate (healing)	
5261						40-60		Slight	Moderate (healing)	
5340	O.		Entire 28 days		20	40-30	84	Moderate	Moderate	7 in litter; 2 killed at end of lactating period; inorganic P of blood was 5.2 mg.; at end of exper., 3.1 mg.
5341						42-42		Moderate (healing?)	Moderate	
5342						40-40		Moderate	Moderate	
5343						42-50		Slight	Moderate	
5344						40-40		Moderate	Moderate	
5505	G.			Last 14 days	6	20-24	84	Slight	Slight	8 in litter
5506						20-30		No	Very slight	
5507						22-20		No	Very slight	
5508						22-24		Slight	Very slight	
5501						20-28		Moderate	Moderate	
5502						20-28		Moderate	Moderate	
5503						20-32		Moderate	Moderate	
5504						22-30		Moderate	Moderate	

* No. 84 diet is Sherman-Pappenheimer low phosphorus, high calcium dietary. In the dry milk dietary, 5 per cent. of the flour was replaced by an equivalent amount of dried milk.

rickets, as has been frequently emphasized, but also to the prerachitic stage. For example, in our experience, animals that have grown exceedingly rapidly during the period preceding the test subsequently develop rickets more readily than animals that have grown but slightly or moderately—the result of a storing in the tissues of the animals in which growth has been tardy. This phenomenon was recently exemplified by a series of rats which were rendered excessively large by feeding an unusually liberal and varied diet throughout lactation. These rats weighed as much as 60 gm. at the age of 4 weeks, the time when they were weaned and the experimental diet begun. Rats of our stock usually weigh from 35 to 40 gm. at this age. Before the experiment was undertaken, it was thought that these exceptionally well nourished animals might prove resistant to rickets. As a matter of fact, they evinced no refractory tendency whatsoever, having apparently no reserve of the antirachitic factor on which to draw. In contradistinction to these animals, it will be noted, as shown in the accompanying table, that the rats that were fed cod liver oil during the lactating period, and were protected thereby, were not large when they were weaned and placed on the rickets-producing dietary. The protection afforded by cod liver oil must be regarded not as an absolute factor but as one which bears a reciprocal relationship to the growth of the body. Its active principle is stored only when the oil has been taken in quantities exceeding the immediate demands of the body, and, on the other hand, is entirely consumed when the growth ratio has been excessive.

These experiments are not to be interpreted as indicating that the subsequent development of rickets in the young is not influenced by the diet of the mother during the period of pregnancy or lactation. Experiments recently published from this laboratory show that such was not the case.[2] Tests of this nature involving, necessarily, two generations are most complex, depending on various nutritional factors. An adequate discussion of this subject would lead too far afield and will be postponed until other aspects are considered.

Korenchevsky,[3] who has carried out a large number of experiments in this field of investigation, came to the conclusion that cod liver oil was of antirachitic value when given to the mother during pregnancy or lactation. It is difficult to compare the two series of experiments as he gave calcium in addition to cod liver oil. Furthermore, the nutritional condition of the animals, depending on the stock, on the nature of the standard dietaries and on other factors, renders it impossible to correlate experiments of this nature.

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2. Hess, A. F.; Weinstock, Mildred, and Tolstoi, E.: J. Biol. Chem. **57,** No. 3, 1923.

3. Korenchevsky, V.: Medical Research Council, Special Report, No. 71, 1922.

SUMMARY

Our experiments indicate that cod liver oil, when given to the mother during pregnancy, cannot be relied on to protect the offspring from rickets. Furthermore, the active principle of the oil is not excreted in the milk in adequate amount; rats receiving even large doses of cod liver oil were unable to afford protection to their young, which were subsequently placed on rickets-producing diets.

On the other hand, cod liver oil did confer subsequent protection when fed to the young during the latter half of the lactating period directly and not through the medium of the mother's milk, demonstrating the essential difference between direct and indirect nutrition. This protective power suggests that the specific factor in the oil can be stored by the young and drawn on in time of need. Although it cannot be taken for granted that these results are applicable to human rickets, it is probable that such distinction as exists is one of degree rather than of kind.

BLOOD PRESSURE IN THE NEW-BORN *

M. PIERCE RUCKER, M.D.
AND
J. W. CONNELL, M.D.
RICHMOND, VA.

Physiology and pathology approach each other so closely in obstetrics that it is often difficult to distinguish the one from the other. In fact, there are two groups of obstetricians, the one typified by Eden,[1] who considers pregnancy and parturition essentially physiologic; the other, by DeLee,[2] who considers all labor abnormal. Between these extremes, there is a large group that considers the pregnant woman to be just on the dividing line between the normal and the abnormal, usually on the normal side, but often, with the least provocation, getting over the line into the abnormal.

This condition of affairs is equally true of the new born, as one readily realizes in studying the causes of death of infants at birth and shortly thereafter. In Williams' classic study of infant deaths,[3] the next to the largest class of etiologic factors was the unknown (18 per cent.). If to these there be added other groups in which necropsy revealed no definite cause for death, i. e., prematurity (7.1 per cent.), inanition (3.3 per cent.) and debility (0.7 per cent.), we have a group that comprises 29.1 per cent. of the stillbirths and neonatal deaths, which is larger by 2.5 per cent. than Williams' first group. Thus, it will be seen that, in almost 30 per cent. of his 705 deaths, necropsy failed to reveal a satisfactory explanation. Holt and Babbitt,[4] in a study of 10,000 deliveries in the Sloane Hospital for Women in New York, found that 58 per cent. of the deaths of infants in the first two weeks were due to congenital weakness and atelectasis. "Except for the larger rôle played by syphilis, the causes of stillbirth in no way differ from those which produce death during the first days of life." Of 200 stillbirths and neonatal deaths at the Royal Maternity Hospital at Edinburgh, Browne [5] found that 40 per cent. were due to asphyxia.

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* Received for publication, Oct. 31, 1923.

1. Eden, T. W.: Maternal Mortality of Child-Bearing, Lancet **2**:998, (Nov. 11) 1922.
2. DeLee, J. B.: Prophylactic Forceps Operation, Am. J. Obst. & Gynec. **1**:34 (Oct.) 1920.
3. Williams, J. W.: Limitations and Possibilities of Prenatal Care, J. A. M. A. **64**:95 (Jan. 9) 1915.
4. Holt, L. E., and Babbitt, Ellen C.: Institutional Mortality of the New-Born, J. A. M. A. **64**:287 (Jan. 23) 1915.
5. Browne, F. J.: Still-Birth: Its Causes, Pathology and Prevention, Edinburgh M. J. **27**:153, 199 and 286, 1921.

When the cause of death is determined largely on clinical data, as in Moran's[6] series or in the Mortuary Reports, the inanition and unknown class is even larger. On the other hand, had the newer methods of opening the cranium, such as that of Beneke,[7] the importance of which is so stressed by Ehrenfest,[8] been used, it is possible that the unknown class of Williams and Holt and Babbitt might have been smaller. Even with this possible deduction, it is evident that what we might term "physiologic" death, or death with no discoverable pathologic condition, ranks high among the causes of infant mortality.

The magnitude of the question of infant mortality was pointed out by Foote[9] in a recent editorial. "It is appalling, therefore, to realize that probably 8 per cent. of the 2,500,000 children born annually in the United States will pass away within a month from the time that they are born." If a large percentage of these 200,000 deaths are physiologic, or at least have no discoverable cause, then any investigation of the physiology of the new-born should be of interest, not only to the physiologist, but also to the pathologist, the obstetrician, the pediatrician and the public health worker.

THE LITERATURE

What is the normal blood pressure in new-born infants? This question presented itself to one of us in connection with a group of stillbirths that had rather a characteristic symptomatology. These infants were born, or were delivered, with considerable difficulty. They were pale and limp, and were apparently dead, except for pulsations at the apex, which continued for thirty or forty minutes. There was no obstruction in the air passages, but no respiratory movements could be started by any of the usual methods of resuscitation. Necropsy showed that our artificial respiration had inflated the lungs, at least partially, and there was no discoverable cause for death.

The question whether shock played a part in these cases at once brought up the question of blood pressure. Whatever may be the actual cause of shock; whether it be due to vasomotor exhaustion, as was suggested by W. W. Keen, S. Weir Mitchell and G. R. Moorehouse;[10] to mental emotion and bodily pain, singly or in combination;[11]

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6. Moran, J. F.: Endowment of Childhood from the Obstetric Standpoint, J. A. M. A. **65**:2224 (Dec. 25) 1915.

7. Beneke: Ueber Tentoriumzerreissungen bei Geburt sowie die Bedeutung der Duraspannung für chronische Gehirnerkrankungen, München. med. Wchnschr. **57**:2125, 1910.

8. Ehrenfest, Hugo: Birth Injuries of the Child, New York and London, D. Appleton and Company, 1922, p. 27.

9. Foote, John: The Hazardous First Month, Mother and Child **4**:28, 1923.

10. Circular No. 6, Surgeon-General's Office, 1864.

11. Mansell-Moullin, C. W.: International Encyclopaedia of Surgery, Revised Ed., New York, William Wood & Co., 1889, Vol. 1, p. 361.

to a reflex inhibition of the centers of the cord;[12] to fat emboli;[13] to acidosis;[14] to epinephrin exhaustion;[15] to a toxic agent developed in damaged tissue;[16] to damaged brain cells;[17] or to one of many different causes, as suggested by Mann,[18] extremely low blood pressure is the constant finding. In fact, Erlanger[19] defines "shock" as a reduction of arterial pressure to or below 50 mm. of mercury, together with other usual signs.

No one with whom we came in contact, obstetrician, pediatrician, or physiologist, could tell us what the blood pressure for the new-born should be under normal conditions, or how to obtain it in such cases as we were interested in at the time; although there have been quite a number of studies of the arterial pressure in older children, in both health and disease. Attempts to make clinical use of sphygmomanometry in children begin with Eckert[20] and von Basch.[21] Eckert measured the pressure in the temporal arteries, while von Basch used the radial. The method they employed was compression of the artery with a solid button measuring the force necessary to occlude the vessel. Seiler[22] used a Sahli modification of the von Basch instrument and compared his results with those he obtained with the Riva-Rocci sphygmomanometer, which had by this time come into general use. His results were almost identical with the two instruments. Friedemann[23] studied

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12. Meltzer, S. J.: Nature of Shock, Arch. Int. Med. **1**:571 (July 15) 1908.

13. Park, Rosewell: On Fat Embolism, New York M. J. **40**:177, 1884; Bissell, W. W.: The Amount of Fat in the Blood Stream of Persons with Broken Bones, J. A. M. A. **67**:1926 (Dec. 23) 1916; Porter, W. T.: Fat Embolism, a Cause of Shock, Boston M. & S. J. **176**:248 (Feb. 15) 1917; Porter, W. T.: Shock, New York M. J. **106**:894 (Nov. 10) 1917.

14. Henderson, Yandell; Prince, A. L., and Haggard, H. W.: Observations on Surgical Shock, J. A. M. A. **69**:965 (Sept. 22) 1917.

15. Corbett, J. F.: Suprarenal Gland in Shock, J. A. M. A. **65**:380 (July 31) 1915.

16. Cannon, W. B.: Studies in Experimental Traumatic Shock, Arch. Surg. **4**:1 (Jan.) 1922.

17. Pike, F. H., and Coombs, Helen C.: The Relation of Low Blood Pressure to a Fatal Termination in Traumatic Shock, J. A. M. A. **68**:1892 (June 23) 1917; Crile, G. W.: The Mechanism of Shock and Exhaustion, J. A. M. A. **76**: 149 (Jan. 15) 1921.

18. Mann, F. C.: Further Experimental Study of Surgical Shock, J. A. M. A. **71**:1184 (Oct. 12) 1918.

19. Erlanger, Joseph; Gesell, Robert; Gasser, H. S., and Elliott, B. L.: An Experimental Study of Surgical Shock, J. A. M. A. **69**:2089 (Dec. 22) 1917.

20. Eckert, Alexandrine: Zeitung Wratsch., 1882, quoted by Fr. Arnheim, Ztschr. f. klin. Med. **5**:376, 1882.

21. Von Basch, S.: Ueber Pulse Fühlen, Pulse Schreiben, und Pulse Messen, Arch. f. Kinderh. **5**:277, 1884.

22. Seiler, H.: Praktische Bemerkungen über den Blutdruck und über Normalwerte des Blutdruckes im Kindesalter, Cor. Bl. f. schweiz. Aerzte **40**:433, 1910.

23. Friedemann, J. H.: Blutdruckmessungen bei Diphtherie, Jahrb. f. Kinderh. **36**, 1893.

a few fatal cases of diphtheria with the von Basch apparatus. Shaw,[24] Heim,[25] Kolassawa,[26] Neu[27] and Eminet[28] made use of Gärtner's tonometer.[29] Hensen,[30] Biernacki,[31] Cook,[32] Beretta,[33] Oppenheimer and Bauchwitz,[34] Trumpp,[35] Stowell,[36] Wolfensohn-Kriss,[37] Gordon,[38] Michael,[39] Salle,[40] Kaupe,[41] Melvin and Murray,[42] and Seitz and Becker[43] used some form of the Riva-Rocci sphygmomanometer. Leitao,[44] in a comprehensive study of both artificially fed and breast-fed babies, used the Erlanger and Hooker apparatus.[45] Hill[46] used a Nicholson sphygmomanometer with the ordinary arm band folded once

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24. Shaw, H. L. K.: Arterial Tension in Childhood, Albany M. Ann. **21**:88, 1900.

25. Heim, P.: Das Verhalten des Blutdruckes bei neuropathischen Kindern, Deutsch. med. Wchnschr. **26**:320, 1900.

26. Kolassawa, Anna: La pression sanguine chez les enfants dans les conditions physiologiques et pathologiques, Arch. de méd. d. enf. **5**:421, 1902.

27. Neu, Maximilian: Experimentelle und klinische Blutdruck unter suchungen mit Gärtners Tonometer, Heidelberg, 1902.

28. Eminet, P. P.: Sphygmographie und Tonometrie bei gesunden Kindern im Alter von 7-15 Jahren, Arch. f. Kinderh. **46**:328, 1907.

29. Norris, G. W.: Blood-Pressure: Its Clinical Applications, Ed. 2, Philadelphia and New York, Lea & Febiger, 1916, p. 76.

30. Hensen, H.: Beiträge zur Physiologie und Pathologie des Blutdrucks, Deutsch. Arch. f. klin. Med. **67**:442, 1900.

31. Biernacki, J.: The Essential Toxic Symptoms of Diphtheria, Brit. M. J. **2**:1787, 1899.

32. Cook, H. W.: Clinical Value of Blood-Pressure Determinations as a Guide to Stimulation in Sick Children, Am. J. Med. Sc. **125**:433, 1903.

33. Beretta, Linita: Della tensions arteriosa nei bambini sani nelle loro diverse eta., Clin. med. ital. **42**:100, 1903.

34. Oppenheimer, K., and Bauchwitz, S.: Ueber den Blutdruck bei gesunden Kindern, Arch. f. Kinderh. **43**:415, 1905.

35. Trumpp, J.: Blutdruckmessungen an gesunden und kranken Säuglingen, Jahrb. f. Kinderh. **63**:43, 1906.

36. Stowell, W. L.: Blood-Pressure in Children, Arch. Pediat. **25**:88, 1908.

37. Wolfensohn-Kriss, Fr. P.: Ueber den Blutdruck im Kindesalter, Arch. f. Kinderh. **53**:332, 1910.

38. Gordon, Lennox: The Blood Pressure in Cases of Acute Nephritis of Children, Arch. Pediat. **28**:343, 1911.

39. Michael, May: A Study of Blood-Pressure in Normal Children, Am. J. Dis. Child. **1**:272 (April) 1911.

40. Salle, V.: Ueber Blutdruck im Klindesalter, Jahrb. f. Kinderh. **73**:273, 1911.

41. Kaupe, Walter: Der Blutdruck im Kindesalter, Monatschr. f. Kinderh. **9**:257, 1910.

42. Melvin, G. S., and Murray, J. R.: Blood Pressure Estimation in Children, Brit. M. J. **1**:669, 1915.

43. Seitz, A., and Becker, Felix: Ueber den Blutdruck beim Neugeborenen, Zentralbl. f. Gynäk. **44**:1338, 1920.

44. Leitao, Mello: Pressae arterial na infancia, Arch. brasil. de med. **1**: 729, 1911.

45. Erlanger, Joseph: A New Instrument for Determining the Minimum and Maximum Blood Pressures in Man, Johns Hopkins Hosp. Rep. **12**:53, 1904.

46. Hill, H. K.: A Note on Blood Pressure in Infants, Especially in Gastro-enteritis, Arch. Pediat. **30**:588, 1913.

upon itself and applied to the thigh; while Judson and Nicholson [47] used a modification of the Fedde [48] pith ball attachment to make the pulsations of the artery more apparent. With a second cuff on the forearm, or leg, as the case might be, they were able to make both systolic and diastolic readings. Without this attachment, they found it very difficult to take blood pressure readings in infants. Hoobler [49] is the only one who has published graphic records of children's blood pressure. He found that readings taken on the thigh were from 10 to 15 mm. of mercury higher than those taken on the arm. The French observers Gaujoux,[50] Jarricot,[51] Koessler [52] and Balard [53] employed the sphygmometric oscillometer of Pachon.[54] Morse [55] used a Tycos sphygmomanometer.

The results have varied widely, and those investigators, Biernacki,[31] Cook,[32] Heim,[25] Kolossawa,[26] Hoobler,[56] Hill,[46] Morse and Hassmann,[55] Howland and Hoobler,[57] Gordon [38] and Hill,[46] who were interested in the blood pressure in connection with some pathologic condition of children, have felt the need of determining a normal for themselves. In this connection, Utheim [58] states: "A survey of the literature in regard to blood pressure in infancy reveals rather widely varying values for normal infants. The reasons for this are, as usual, that the methods employed have been different, examinations have been few

47. Judson, C. F., and Nicholson, Percival: Blood-Pressure in Normal Children, Am. J. Dis. Child. **8**:257 (Oct.) 1914.

48. Fedde, N.: A Pith-Ball Manometer, Med. Rec. **78**:105, 1910.

49. Hoobler, B. R.: An Automatic Device for Reading Systolic and Diastolic Blood Pressures, Med. Rec. 80:1323, 1911; The Standardization of Blood Pressure Readings by Means of an Automatic Device for Indicating Systolic and Diastolic Pressures in Children, Am. J. Dis. Child. **4**:46 (July) 1912.

50. Gaujoux, E.: Recherches sur la tension artérielle normale chez l'enfant, Ann. de méd. et chir. inf. **12**:435, 1908.

51. Jarricot: Measure de la tension artérielle chez les enfants, Pediatrie Practique **10**:392, 1912.

52. Koessler, L.: D'oscillométrie appliquée à l'etude de la tension artérielle chez les enfants, Thèse de Paris, 1912.

53. Balard, Paul: Modifications évolutives du pouls et de la tension artérielle chez le nouveau-né dans les premier jours de la vie, étudiées par l'oscillométrie, Compt. rend. Soc. de biol. **73**:483, 1912; La tension artérielle et l'oscillométrie chez le Nouveau-Né, Gaz. d. hôp. **86**:837, 1913.

54. Pachon, V.: La mesure de la pression artérielle par la méthode des oscillations, Paris méd., 1911-1912, p. 122.

55. Morse, J. L., and Hassmann, D. M.: Effect of Cold Air on the Blood Pressure in Pneumonia, Am. J. Dis. Child. **12**:445 (Nov.) 1916.

56. Hoobler, B. R.: The Effects of Cold Air on Blood Pressure in Children and Young Adults in Various Stages of Tuberculosis, Am. J. Dis. Child. **4**:307 (Nov.) 1912.

57. Howland, John, and Hoobler, B. R.: The Effect of Cold Fresh Air on the Blood Pressure in Pneumonia of Children, Am. J. Dis. Child. **3**:294 (May) 1912.

58. Utheim, Kirsten: A Study of the Blood and Its Circulation in Normal Infants and in Infants Suffering from Chronic Nutritional Disturbances, Am. J. Dis. Child. **20**:366 (Nov.) 1920.

and the selection of material has not been sufficiently careful." She adds a table for normal children of ages from one half to 28 months, based on thirty-five cases.

The question of width of the cuff is one that was stressed by Recklinghausen.[59] In the work on children, various widths have been used, varying from 5 to 13 cm. Oppenheimer and Bauchwitz [34] compared their figures with those of Eckert and Beretta. Although Eckert used an instrument of an entirely different type, her figures are very similar to those of Oppenheimer and Bauchwitz. On the other hand, Beretta who used the same kind of instrument but a wider cuff, obtained much lower results; which Oppenheimer and Bauchwitz believe is due to the difference in the width of the cuffs. Judson and Nicholson, however, found no difference in children under 8 years when cuffs of different widths were used. Wolfensohn-Kriss,[37] working in Sahli's clinic, found that the blood pressure (she recorded only the systolic) varied according to age, weight and height. She found no difference between the sexes. Salle found a variation commensurate with the age, and to a certain extent with the weight and height. In his cases, nephritic and neuropathic children had higher pressures. Michael [39] studied 128 normal children, ranging in height from 3½ to 5 feet. The systolic pressure varied from 99 to 125 mm. of mercury. She calculated the pressure in relation to the width of the arm band and the circumference of the arm, as suggested by Sallom;[60] in other words, she figured the pressure according to the number of square millimeters of surface compressed, and found that there was practically no variation in children of different heights. Faber and James,[61] in a study of 651 boys and 450 girls, found that systolic, diastolic and pulse pressures averaged the same for the two sexes in the various age groups, but that the standard of deviation was greater in the girls; that is to say, there was greater variability of all three pressures in females.

Most of the work that has appeared in the literature has dealt with children of from 6 months to 14 years of age. Trumpp [35] reports a few observations on infants with icterus neonatorum. At first, the pressure was 62. It rose to 80 in the first two days of the jaundice, and then fell to 50. His youngest patient was 2 days old when the first observation was made.

Vierordt,[62] in 1855, found the blood pressure in the larger arteries of the new-born to be 111. Neu,[27] in his inaugural dissertation, reports

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59. Recklinghausen, H.: Unblütige Blutdruckmessung, Arch. f. exper. Path. u. Pharmakol. **55**:375, 412, 1906.

60. Sallom, A. K.: Standardization of Blood Pressure, New York M. J. **92**:620, 1910.

61. Faber, H. K., and James, C. A.: The Range and Distribution of Blood Pressure in Normal Children, Am. J. Dis. Child. **22**:7 (July) 1921.

62. Vierordt: Daten und Tabellen, Ed. 2, Jena, 1893.

observations with Gärtner's tonometer on a few new-born babies. He found the pressure to be 90. Seitz and Becker [43] studied 102 new-born babies with the Riva-Rocci sphygmomanometer and the palpatory method. In only four cases were they able to obtain the pressure satisfactorily on the first day. *A priori,* they believe that the pressure should be low in infants at birth. All the factors directly concerned, feeble heart action, low peripheral resistance and small blood volume, are present in very young infants. The blood volume of the new-born is one nineteenth of its body weight, in contrast with one thirteenth in the adult. The results that they obtained in the four cases are shown in Table 1.

The most comprehensive study of blood pressure in the new-born has been made by Balard [53] on forty infants. His observations were made at birth and at each subsequent hour for twelve hours, and then each day for ten days. He also recorded the temperature and pulse at the same time, and the daily weight. His chart shows that there was a steady rise in both systolic and diastolic pressure, while there was a marked drop in both the temperature and pulse in the first hours

TABLE 1.—*Blood Pressure of Young Infants (Seitz and Becker)*

Case	1st Day	3d Day	5th Day	7th Day	10th Day
A.....	45	55	55	60	62
B.....	48	60	62	60	76
C.....	..	54	60	63	68
D.....	45	52	52	58	63

of life. The minimum was reached in the seventh hour, after which time there was a rise in both temperature and pulse, but not so rapid as the fall. In 1921, Balard [63] again reported forty cases and reproduced the same chart. He believes that the oscillatory method is the only practical one for very young subjects. Failure to recognize this accounts for the fact that there have been so few studies of the arterial pressure in the new-born. He quotes Mensi as having found the systolic pressure for new-born jaundiced babies to be 85 and Popoff, with the Riva-Rocci instrument, to have found the systolic pressure 70 and the diastolic 40, for babies born at term. He prefers to make his observations on the arm, because the pulsations in the brachial artery are larger and the shape of the arm is more suitable for the application of the cuff. The rubber bag in his arm band measured 6 by 13 cm.

METHOD

At the beginning of our work, we encountered difficulties that we later found to be the experience of everyone who has attempted to

63. Balard, Paul: La tension artérielle et l'oscillométrie chez le nouveau-né, Nourrisson **9**:304 (Sept.) 1921.

take the blood pressures of very young infants. It might be said that we had practically completed our observations before we found any literature on the subject, so that our results are entirely unbiased by any previous work. It is practically impossible to feel a new-born baby's pulse at the wrist. Seitz and Becker [43] were able to obtain it in only four out of 102 cases. While the auscultatory method is applicable in more cases, even this is uncertain and often impossible in very small infants. Hill [46] was unable to obtain readings on several infants even as old as 5 months. Hoobler [49] found it difficult to obtain blood pressure readings in children with the customary apparatus, and devised a special attachment to overcome this difficulty. He used both the arm and the thigh, and found the thigh readings to be from 10 to 15 mm. of mercury higher than those for the arm. Hensen [30] also noted that the thigh gave higher readings. Balard thinks that the oscillatory method is the only

TABLE 2.—*Variations in Mercury Column and Sphygmomanometer Readings*

Mercury Column	Tycos Sphygmomanometer
20	22
30	32
40	42
50	52
60	61
70	70
80	80
90	88
100	99
110	108

one that is clinically applicable to very young infants. In speaking of the instrument that he used, Pachon's oscillometer, Norris [64] classifies it as the most accurate aneroid instrument at present available. He quotes Bachmann's figures to show that in adults the systolic pressure is higher by 20 or 30 mm. when read by the Pachon instrument than when determined by feeling the pulse at the wrist. The oscillometer is very delicate, and with it Balard [65] has been able to demonstrate pulsations in apparently dead babies whose systolic blood pressure was only 10 mm. He has been successful in taking blood pressures in an infant that weighed only 950 gm.

For our work, we have used the arm entirely. The arm band was made by sewing a Barnes bag 4 cm. wide between two layers of sateen in imitation, on a reduced scale, of the arm band supplied with the Tycos sphygmomanometer. The two openings were obtained by putting a Y-tube in the stem of the Barnes bag. An effort was made to show the pulsations of the brachial artery with a Marey tambour and a

64. Norris, G. W.: Blood Pressure, Ed. 2, Philadelphia, Lea & Febiger, 1916, p. 108.

65. Balard: Paul: L'oscillométrie de Pachon, critère de la persistance de la circulation dans la mort apparente du nouveau-né, Presse méd. **21**:252, 1913.

kymograph, but the amplitude of the pulse wave was too small and the inertia of the apparatus was too great. We obtained only a downward-sloping straight line as the pressure in the cuff was slowly released. However, the needle of the Tycos sphygmomanometer showed the pulsations plainly, especially the instrument with the 6 inch dial. Schrump and Zabel [66] emphasize the importance of applying the arm band snugly, and with the small capacity of the bag that we have used this is very necessary. It is quite important that the babies be quiet, as muscular contractions of the arm obscure the pulsation of the artery. Any effort to hold the arm still is resisted, and this makes it difficult to obtain an auscultatory reading. The arm band is adjusted to the bare arm and the baby is covered with a blanket. If he does not become quiet in a few minutes, he is given a bottle of sterile water, which usually appeases him. A few observations were made with the baby at the breast. The readings were repeated several times, and whenever it was possible readings were also made by the auscultatory method. The first distinct pulsation of the needle was taken to indicate the systolic pressure, and the point at which the amplitude of the pulsations diminished, the diastolic pressure. The same instrument was used in all the observations. It was checked against a mercury column with the results shown in Table 2.

MATERIAL

The material was supplied by the wards of the Memorial and St. Philip's hospitals, and for permission to use it in this study we are indebted to Professor Baughman. Supplementary material was obtained from the private practice of one of us. About one third of the subjects were negroes. Roughly speaking, the cases fall into two equal groups, which were studied independently by one or the other of us. Neither of us knew the other's results until the work was practically complete. There was no appreciable difference in the two groups. In order to make the comparison of the oscillatory and the auscultatory methods more complete, observations were made on a few older infants in the baby wards.

ANALYSIS OF RESULTS

Our results are shown in detail in Tables 3 to 6 and the accompanying charts. Babies of mothers with hypertension or toxemia were excluded from Tables 5 and 6, and do not figure in the arithmetical means. The two races were separated for two reasons: (1) we wished to see if there were any difference in the blood pressures in the newborn of the two races, and (2) the well-known difference in size of

66. Schrump, P., and Zabel, B.: Ueber die auskultatorische Blutdruckmessung, München. med. Wchnschr. **56**:704, 1909.

white and negro babies at term presented an insurmountable difficulty in arrangement of tables when the cases were arranged in the order of their maturity as indicated by the total length in centimeters. Table 4 is a comparison of the oscillatory method with the auscultatory method for different days of early infancy. The oscillatory readings are placed above the auscultatory results in the appropriate columns. It will be noted that the findings by the auscultatory method are generally a few millimeters of mercury lower, and that often there is a little uncertainty about the results, as we have indicated by "?".

The arithmetical averages for the fifty-two normal infants are as follows: first day, 55/40; second day, 58/41; third day, 54/39; fourth day, 58/42; fifth day, 60/42; sixth day, 63/43; seventh day, 61/44; eighth day, 63/44; ninth day, 64/43, and tenth day, 64/41. This is

TABLE 3.—*Systolic Blood Pressure in Infants*

	Age											
	Days								Years			
Observers	1	2	3	4	5	10	15	30	½	1	2	4
Seitz and Becker	43	48	55	56	58	66	70	82	..	..	..	..
Balard	55	60	60	65	65	75	..	..	..	..	..	..
Rucker and Connell	55	58	54	58	60	64	..	..	..	..	..	..
Beretta	..	65	..	58	..	61	72	75	..	..	..	..
Trumpp	..	..	62	..	80	50	..	60	(jaundiced babies)			
Koessler	..	..	..	..	70	..	..	..	..	..	..	..
Utheim	..	..	..	..	..	..	80	85	90	100	100	..
Leitao	..	..	..	..	..	..	..	66	73	80	87	99
Cook	..	..	..	..	..	..	..	70	82	..	..	..
Oppenehimer and Bauchwitz	..	..	..	..	..	..	..	..	80	90	90	..
Gordon	..	..	..	..	..	..	..	..	71	73	80	83
Kolassawa	..	..	..	..	..	..	..	..	..	80	..	..
Eckert	..	..	..	..	..	..	..	..	..	..	97	100
Seiler	..	..	..	..	..	..	..	..	..	..	80	83
Stowell	..	..	..	..	..	..	..	..	..	..	..	89
Kaupe	..	..	..	..	..	..	..	..	..	..	..	86
Wolfensohn-Kriss	..	..	..	..	..	..	..	..	..	..	..	85
Judson and Nicholson	..	..	..	..	..	..	..	..	..	..	..	91

shown graphically by Chart 1, which is that of a representative case, and by Chart 2, which is a composite chart based on the foregoing averages. These figures are somewhat higher than those of Seitz and Becker, and a little lower than Balard's, as Table 3 shows.

The cases that we have grouped as abnormal are interesting, although they are too few to warrant any conclusions.

REPORT OF CASES

CASE 1 (Chart 3).—A boy, white, 47 cm. long and weighing 2,270 gm., who was delivered by breech extraction after administration of pituitary extract had proved ineffectual, was apparently normal at birth. On the third day, he had a shrill cry, twitching of the eyelids, deviation of the eyes to the left and a rotation of the head strongly to the left. The child refused to nurse. Kernig's sign was absent. Lumbar puncture was refused. The child died the morning of the fourth day. Necropsy was not obtainable. We postulate this as a case of intracranial hemorrhage. The blood pressure, systolic and diastolic, for the first three days of life was 54, 46; 54, 46, and 88, 42, respectively.

TABLE 4.—*Comparison of Oscillatory and Auscultatory Methods**

Name	Length, Cm.	Weight, Gm.	Sex†	Race	Days 1	2	3	4	5	6	7	8	9	10
1. J. P.	47	2,440	♀	W	.....	.....	54/42 50/40	.....	.....	.....	.....	.....	.....	.....
2. W. W.	47	2,724	♀	C	52/22 50/24	56/24 52/30	58/38 54/36	54/38 50/38	60/40 56/40	.....	.....	.....	.....	.....
3. M. C.	49	3,248	♀	C	52/43 50/39	54/40 52/38	56/40 57/ ?	.....	56/44 55/39	.....	.....	.....	.....	.....
4. V. C.‡	49	3,248	♀	C	60/39 59/39	63/42 64/39	65/44 62/42	.....	.....	.....	.....	.....	70/45 68/41	66/41 62/38
5. L. P.	49	3,178	♀	C	52/39 52/43	56/39 58/41	52/38 54/ ?	55/44 52/39	61/50 54/42	63/44 56/40	62/39 60/41	68/44 64/40	64/40 60/38	67/41 65/43
6. M. M.	51	4,254	♂	C	52/39 51/ ?	52/37 51/38	52/37 48/ ?	54/40 52/39	.....	57/50 54/45	.....	.....	60/40 56/ ?	.....
7. J. J.	53	3,915	♂	C	56/40 53/ ?	60/43 56/ ?	.....	54/39 50/38	56/39 51/ ?	.....	.....	57/41 51/ ?	.....	.....
8. H. W.	54	4,483	♂	C	56/36 54/39	.....	.....	56/42 56/40	.....	.....	.....	.....	.....	.....
9. P. M.	60	5,641	♂	W	64/60 62/40	62/41 63/39	64/43 62/39	62/40 64/41	.....	61/39 60/40	58/39 62/40	.....	.....	.....

* The oscillatory readings are placed above, the auscultatory below.
† In this column, ♀ indicates female; ♂, male.
‡ Preeclamptic mother.

TABLE 5.—*Blood Pressures of White Infants, Oscillatory Method*

Name	Length, Cm.	Weight, Gm.	Sex	Days 1	1	3	4	5	6	7	8	9	10
Mean	..	.....	..	54/41	61/46	54/43	56/44	61/42	62/44	62/45	63/42	67/46	64/42
1. J. S.	45	2,497	♀	40/32	.....	.....	.....	.....	.....	.....	.....	.....	.....
2. W. S.	46	2,440	♀	55/36	54/36	56/42	61/44	68/44	62/38	58/40	64/44	68/46	65/42
3. L. N.	46	2,270	♀	45/35	65/50	54/46	.....	58/48	.....	64/46	62/48	.....	.....
4. F. S.*	47	2,553	♂	.....	.....	54/44	.....	54/44	.....	.....	.....	.....	.....
5. J. P.	47	2,440	♂	52/44	54/44	54/42	.....	.....	.....	.....	.....	.....	52/40
6. W. B.	47½	2,851	♀	58/48	62/50	.....	.....	.....	.....	54/44	.....	.....	.....
7. I. W.	48	3,403	♂	54/36	.....	55/39	63/44	62/43	64/43	62/49	64/39	.....	.....
8. F. S.*	48	2,837	♂	.....	.....	49/40	.....	44/34	.....	.....	.....	.....	.....
9. I. S.	48	2,851	♀	50/42	48/40	.....	.....	54/41	.....	.....	.....	.....	.....
10. T. T.	49	.....	♂	63/42	61/40	54/40	60/40	62/44	63/41	62/39	67/44	65/38	66/40
11. J. F.	49	2,651	♂	56/44	56/38	52/40	.....	.....	.....	.....	.....	.....	.....
12. I. L.	50	.....	..	46/38	52/40	.....	.....	.....	60/48	.....	78/64	.....	.....
13. G. Y.	50	3,403	♂	52/46	48/36	.....	.....	.....	.....	.....	.....	.....	.....
14. C. J.	50	3,745	♀	64/44	66/46	.....	.....	.....	.....	.....	.....	.....	.....
15. V. P.	50	2,851	♂	54/46	62/44	.....	.....	78/64	.....	.....	.....	.....	.....
16. V. M.	50	3,178	♂	.....	.....	.....	66/48	.....	.....	.....	.....	.....	54/42
17. J. L.	50	4,303	♂	48/30	56/44	61/46	.....	.....	.....	.....	.....	.....	.....
18. C. C.	50½	3,178	♀	52/38	52/40	.....	.....	.....	.....	60/48	.....	.....	78/64
19. P. R.	51	2,894	♂	48/36	53/39	50/32	56/39	.....	.....	61/43	63/41	62/38	64/41
20. S. P.	51	2,837	♀	.....	60/40	.....	60/40	.....	56/46	.....	.....	**78/48**	**62/44**
21. M. C.	51	3,178	♀	56/44	58/44	.....	.....	.....	.....	.....	.....	.....	.....
22. W. W.	51	3,178	♀	54/40	.....	.....	.....	.....	.....	.....	.....	.....	.....
23. G. N.	51	4,086	♀	56/50	.....	.....	.....	78/60	.....	.....	.....	.....	.....
24. R. L.	52	3,415	♂	54/34	.....	.....	.....	.....	58/36	.....	.....	.....	.....
25. C. M.	52	3,745	♂	57/44	56/38	52/30	50/44	56/44	58/40	.....	62/44	61/40	70/50
26. P. S.	52	3,972	♂	58/38	.....	.....	.....	.....	.....	.....	.....	.....	.....
27. V. A.	52	3,178	♀	56/38	58/40	.....	.....	.....	.....	76/64	.....	84/70	.....
28. H. J.	52	3,461	♂	50/42	53/34	.....	.....	.....	.....	56/40	.....	.....	.....
29. J. D.	53	3,518	♂	60/40	62/48	.....	.....	.....	80/66	.....	.....	.....	.....
30. K. M.	53	3,211	♂	72/64	68/54	.....	46/40	.....	.....	.....	.....	.....	.....
31. P. M.†	60	5,646	♂	64/40	62/41	64/43	62/40	.....	61/39	68/39	59/45	60/42	66/44
32. K. C.	..	3,175	♂	.....	.....	.....	52/38	56/48	.....	.....	.....	.....	.....

* Twins. † Cesarean section.

TABLE 6.—*Blood Pressures of Negro Infants, Oscillatory Method*

Name	Length, Cm.	Weight, Gm.	Sex	Days 1	1	3	4	5	6	7	8	9	10
Mean	..	.....	..	55/39	55/39	54/39	57/41	56/40	60/41	60/41	62/43	62/42	64/43
1. I. M.	45	2,270	♂	48/42	50/42	.....	.....	.....	.....	.....	.....	.....	.....
2. W. W.	47	2,724	♀	52/22	56/24	58/38	54/38	60/40	66/44	62/41	61/39	64/41	63/40
3. C. W.	47	3,051	♂	56/36	54/37	58/40	60/41	.....	59/42	60/39	59/44	58/41	60/42
4. H. A.*	47	3,340	♂	58/40	52/39	50/36	49/39	56/40	54/38	52/41	58/44	56/40	60/44
5. D. T.	48	2,497	♂	56/38	54/40	62/38	64/34	66/48	64/44	64/46	70/50	68/46	64/40
6. M. C.	48	2,724	♀	52/43	54/40	55/40	56/40	54/41	56/44	51/44	62/40	63/40	.....
7. H. G.	49	3,340	♀	54/44	58/44	62/44	60/36	64/42	62/38	63/41	68/44	64/41	66/39
8. L. P.†	49	3,178	♀	54/39	56/38	52/38	53/44	61/50	63/44	62/39	68/44	64/40	67/41
9. C. B.	49	2,497	♀	62/41	56/39	58/40	57/40	60/44	62/46	70/40	.....	.....	.....
10. M. G.*	50	.....	♂	60/42	62/40	68/38	.....	.....	.....	68/41	.....	.....	.....
11. B. B.	50	3,440	♀	57/42	57/40	55/40	59/38	58/41	62/42	61/40	66/36	.....	64/44
12. K. D.	50	3,178	♀	56/39	52/36	.....	58/41	59/44	58/42	56/39	60/40	58/38	66/40
13. C. B.	50	3,291	♂	61/40	60/40	58/40	68/41	64/41	70/40	68/40	.....	70/42	72/42
14. A. B.	51	3,802	♂	57/41	58/41	61/42	59/42	62/40	59/44	63/40	64/50	59/41	64/42
15. C. H.	51	4,142	♂	54/38	58/44	52/39	51/40	54/38	56/38	55/41	58/49	57/44	56/45
16. M. M.	51	4,354	♂	52/39	52/37	52/37	52/38	64/40	56/40	57/40	54/38	60/40	62/40
17. G. H.	51	3,178	♀	54/39	55/44	58/38	56/38	57/38	59/41	56/46	.....	.....	.....
18. A. M.	52	.....	♂	51/40	54/39	50/40	56/42	58/39	62/39	61/40	65/36	66/36	70/40
19. J. J.	53	3,915	♂	56/46	60/43	54/40	54/39	56/39	56/41	58/39	67/44	69/44	62/40
20. H. W.	54	4,483	♂	56/36	52/38	48/36	.....	56/42	58/44	60/40	.....	68/48	70/48

* Cesarean section.
† This is the representative case shown on Chart 1.

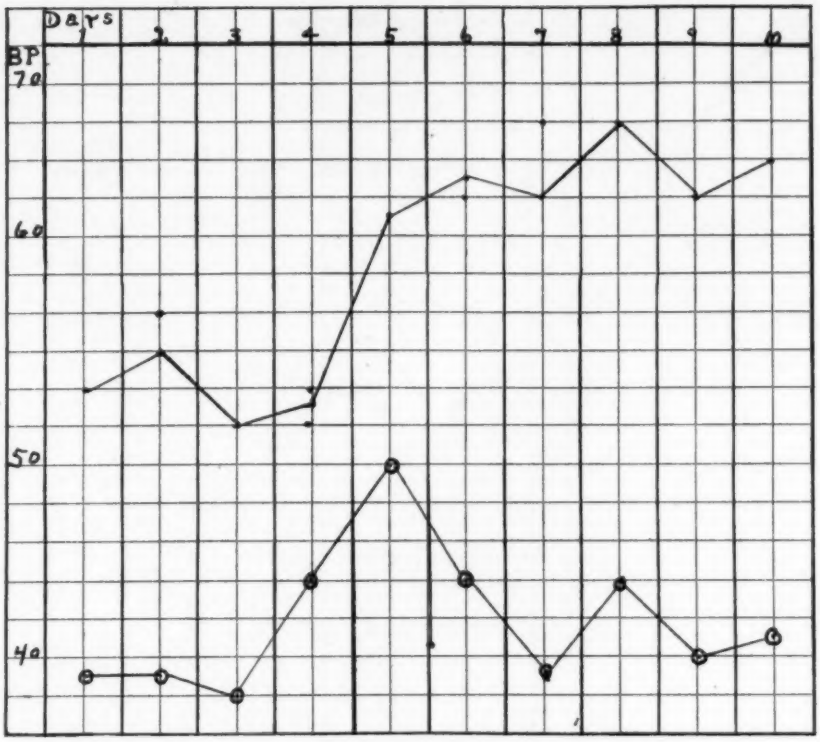

Chart 1 (L. P., colored).—Pressure curves in a representative case. In this chart and the charts following, the upper line represents the systolic, the lower the diastolic blood pressure.

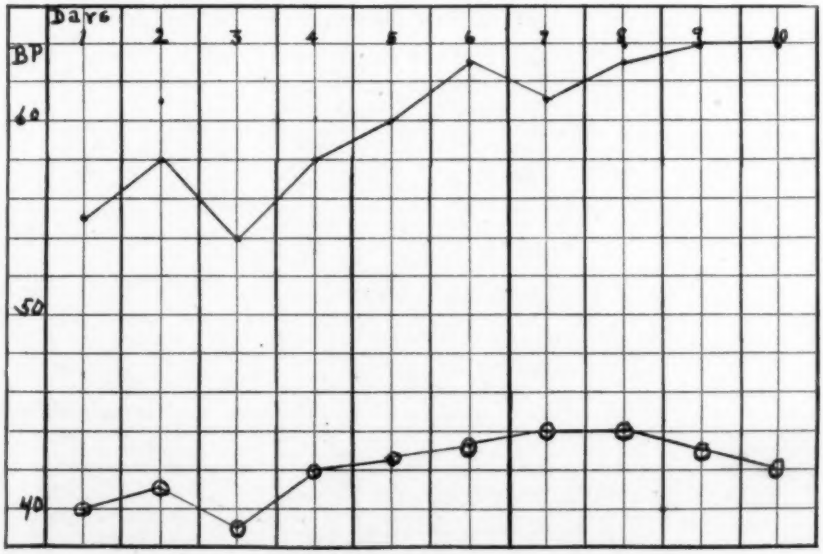

Chart 2.—Composite curves of fifty-two infants.

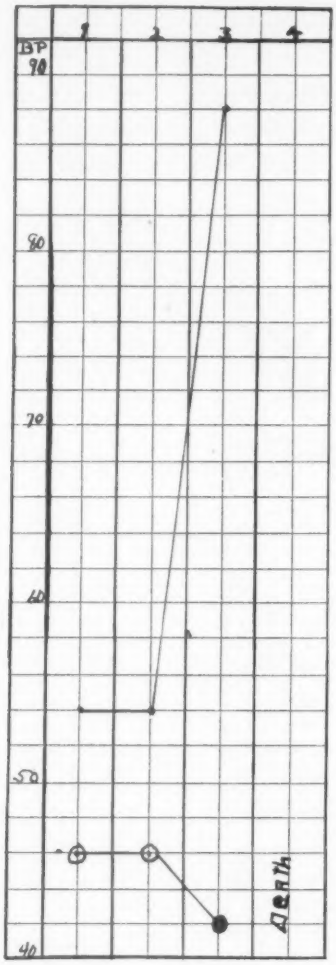

Chart 3 (Case M. W.).—Pressure curves in a case of intracranial hemorrhage (?).

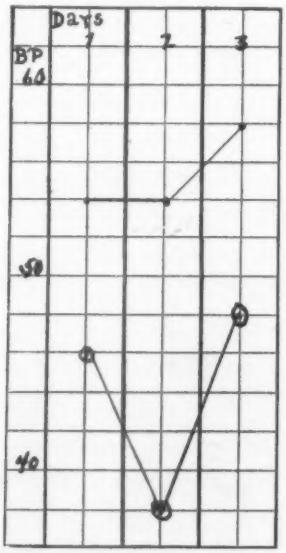

Chart 4 (Case J. W. J.).—Pressure curves in a case of eclampsia.

Case 2 (Chart 4).—A girl, white, 46 cm. long and weighing 2,270 gm., was delivered by version. The mother was in a state of eclampsia. She had three convulsions, on the day of admission to the hospital, which were controlled with morphin, gastric lavage and colonic irrigations. Two days later, her blood pressure rose from 140 systolic and 100 diastolic to 160 systolic and 110 diastolic, and she complained of severe headache. Labor was induced. Postpartum pressure was: systolic, 110; diastolic, 80. Puerperium was eventful. The baby's blood pressure, systolic and diastolic, for three days was: 54, 46; 54, 38, and 58, 48, respectively.

Case 3 (Chart 5).—A boy, white, 48 cm. long and weighing 2,610 gm., was delivered prematurely because the mother had preeclamptic toxemia. The

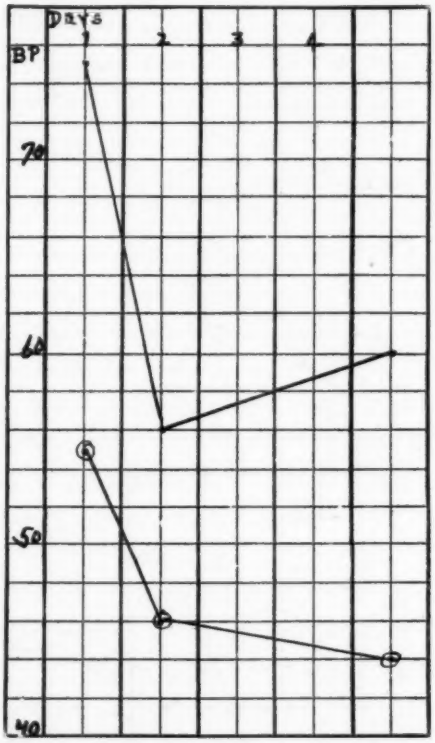

Chart 5 (Case M. P.).—Pressure curves in a case of preeclamptic toxemia.

mother's blood pressure suddenly went from 110 systolic, 60 diastolic to 150 and 100, without any other symptoms. Bread and water diet and rest in bed for a week had no influence on the blood pressure. Six hours after delivery, the blood pressure was 90 and 50. Puerperium was normal. The baby's blood pressure, systolic and diastolic, for the first, second and fifth days was 75, 55; 56, 46, and 60, 44.

Case 4 (Chart 6).—A girl, colored, 46 cm. in length and weighing 2,610 mg., was born of a mother who had been sent to the hospital on account of pernicious vomiting. The mother's blood pressure on admission was 179 systolic, 87 diastolic. Three hours after delivery, it was 158 and 73, and ten days later, 152 and 68. The urine and blood were normal, and the postpartum course uneventful. The baby's blood pressure, systolic and diastolic, for the first ten days was: 61, 35; 64, 36; 60, 39; 65, 43; 68, 43; 62, 42; 60, 43; 65, 44; 62, 40, and 61, 35.

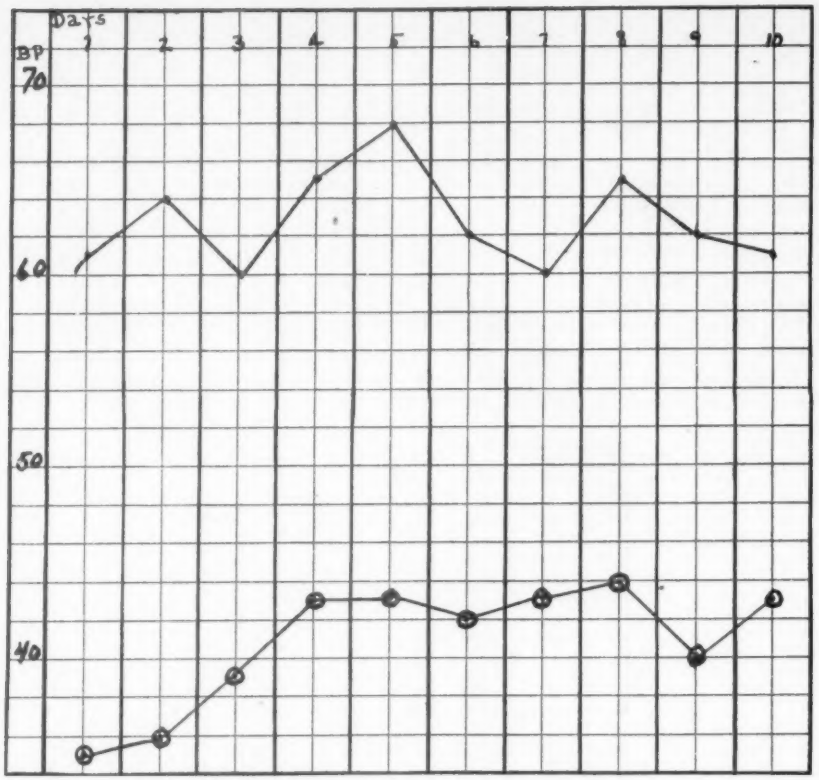

Chart 6 (Case G. F.).—Pressure curves in a case of preeclamptic toxemia.

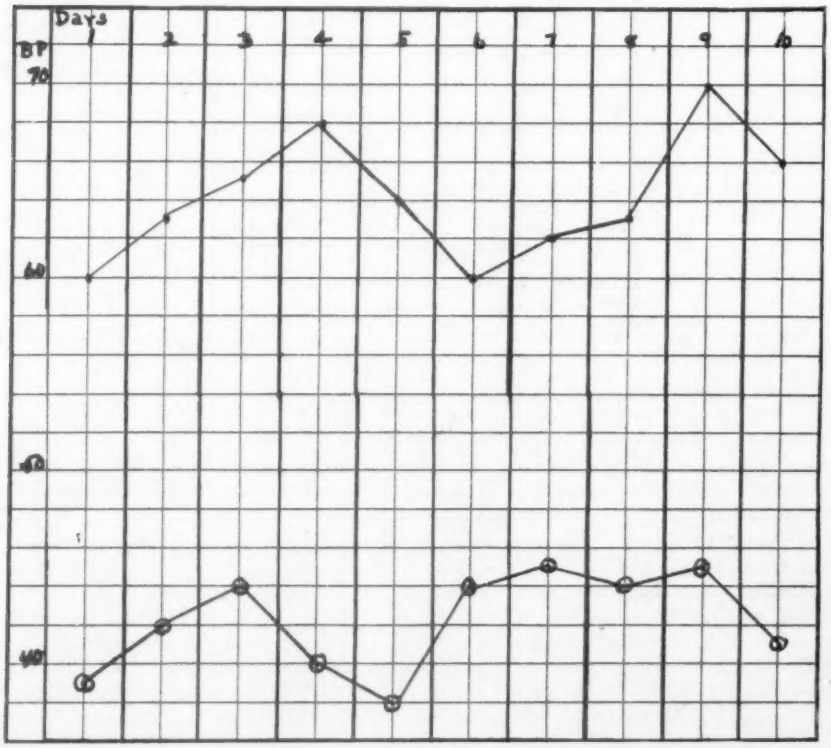

Chart 7 (Case V. C.).—Pressure curves in a case of preeclamptic toxemia.

CASE 5 (Chart 7).—A girl, colored, 49 cm. in length and weighing 3,248 gm., was the result of the seventh pregnancy. The mother was preeclamptic. She was admitted to the hospital at term, but not in labor. Her blood pressure was 172 systolic, 92 diastolic on admission. She complained of headache and had edema of the ankles. Her first four pregnancies ended in abortions at five or six months. The fifth pregnancy was normal, but the sixth resulted in a still-born fetus at term. The urine contained an abundance of albumin, but no casts. Rowntree and Garaghty's test showed an excretion of 55 per cent. in two hours. The blood showed nothing abnormal. The patient remained in the hospital for ten days without going into labor. The headaches were unrelieved, but the edema was reduced somewhat by a restricted diet and rest in bed. Delivery was spontaneous at the end of a labor of twenty minutes. Postpartum blood pressure was 138 systolic, 84 diastolic, and remained at approxi-

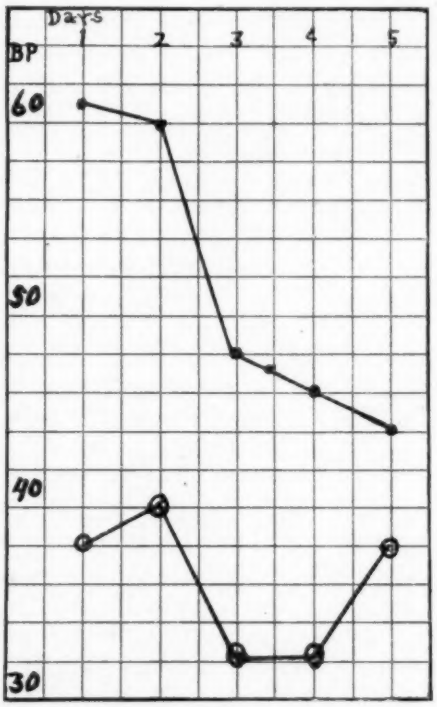

Chart 8.—Pressure curves in case of eclampsia.

mately this point throughout the puerperium. The baby's blood pressure, systolic and diastolic, was: 60, 39; 63, 42; 65, 44; 68, 40; 64, 38; 60, 44; 62, 45; 63, 44; 70, 45, and 66, 41.

CASE 6 (Chart 8).—A white boy, 44 cm. in length and weighing 2,309 gm., was delivered six weeks prematurely. His mother was brought into the hospital in her eleventh convulsion, and she had two more convulsions in rapid succession. Administration of morphin, gastric lavage, colonic irrigations and intravenous glucose solution quickly controlled the convulsions. On the second day after she was admitted, the patient was conscious and it was found that her sight was so impaired that she could not see persons in the room, and could scarcely tell when the light was turned on and off. Her blood pressure, which had dropped from 195 to 135, had again risen to 175. Labor was induced. In

the second stage, she again began to have convulsions. These were controlled with ether and a breech extraction was performed. Twenty-four hours later, a third series of convulsions began, lasting at intervals, when the morphin was discontinued, for four days. The baby was apparently normal, and required no resuscitation. When 2 days old, he had a peculiar crowing cry and a few slight convulsions. His blood pressure, systolic and diastolic, for the first five days was: 61, 38; 60, 40; 48, *32*; 46, *32*, and 44, 38.

The average blood pressure, systolic and diastolic, of the five babies in the cases reported was: 62, 45; 59, 40; 58, 41; 56, 32; 59, 41; 61, 43; 61, 44; 64, 44; 66, 43, and 64, 43. All except one of these babies were premature, and yet their blood pressures after the second day are practically identical with the averages in the normal cases. On the first day the toxic group had decidedly higher pressures. However, the group is too small to warrant our drawing any definite conclusions.

In Case 28 of our series, the patient had hemorrhagic disease of the new-born. On the second day, he vomited bright blood, and had a number of bloody stools. The hemoglobin fell to 42 per cent. He was treated with intramuscular injections, 22 c.c. of whole blood, and made a prompt and satisfactory recovery.

CONCLUSIONS

1. The oscillatory method of taking blood pressure with an aneroid sphygmomanometer is clinically applicable to new-born babies.

2. A comparison with the usual auscultatory method shows that, when used on very young infants, it is sufficiently accurate for clinical purposes.

3. The mean systolic pressure at birth is 55 mm. of mercury. The mean diastolic pressure is 40. The systolic blood pressure increases with the age of the infant more rapidly than does the diastolic pressure.

4. Toxic conditions of the mother seem to have some influence on the blood pressure of the child, which is more marked the first day of life.

5. This method may possibly furnish an additional sign of some value in the diagnosis of intracranial hemorrhage.

6. The blood pressure in general varies directly with the total length of the infant.

7. Sex shows no influence on the blood pressure of the new-born.

8. There is no difference in the blood pressure of the white and of the colored new-born infant.[67]

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67. This article was submitted for publication before Reis and Chaloupka's article "Blood Pressure in Newborn Following Normal and Pathological Labor" (Surg., Gynec. & Obst. **37**:206 [Aug.] 1923) was published. Hence, no reference is made to their work.

BILE PIGMENTS AND BILE SALTS IN THE DUODENAL JUICE OF CHILDREN *

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AND
A. E. GOURDEAU, M.D.
MINNEAPOLIS

Some believe that urobilin is formed only in the intestine. Others believe that, normally, it is also formed in the liver. A reabsorption from the intestine and reexcretion through the liver are both affirmed and denied.[1] It may be considered as proved that increased amounts of urobilin or urobilinogen in the stools, the urine or the duodenal juice are significant either of increased blood destruction or of hepatic disease.[2]

Ylppö examined the stools of fifty infants and found urobilin only after the second week of life.[3] Bauman states that urobilin appears in the intestine of the new-born after the third day.[4]

No reports of urobilin excretion in the duodenal juice of children have hitherto been published, but one of us (R. T.) submitted the finding of urobilinogen (600 dilution units) in the duodenal juice of a child with celiac disease as evidence of hepatic involvement. This opinion was based on Schneider's conclusion that in health more than minimal amounts of urobilinogen do not appear in the duodenal secretion.[5]

The subjects and results of the present work are shown in the accompanying table. The method was that of Wilbur and Addis as modified by Schneider, and the results are indicated as the number of dilutions, reduced to a scale of 1,000, necessary to cause the disappearance of the characteristic spectroscopic absorption bands.

Neither urobilin nor urobilinogen was found in the duodenal juice of seven infants less than 18 days old. Thirteen estimations on eight older infants showed positive results on seven occasions. There was

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* From the Department of Pediatrics, University of Minnesota Medical School.

* Read at a meeting of the American Pediatric Society, French Lick, Ind., June 1, 1923.

1. Whipple, G. H.: Physiol. Rev. **2**:440-459 (July) 1922.
2. Wilbur, R. L., and Addis, Thomas: Urobilin: Its Clinical Significance, Arch. Int. Med. **13**:235 (Jan.) 1914; Schneider, J. P.: Anemia, Arch. Int. Med. **17**:32-39 (Jan.) 1916.
3. Ylppö: Ztschr. f. Kinderh. **9**:208-318, 1913.
4. Bauman: Endocrinology and Metabolism **3**:1921.
5. Taylor, Rood: Tr. Am. Pediat. Soc. **34**:100-113, 1922.

no uniformity, the pigment being at one time present and at another absent in the same patient. The two patients with celiac disease gave findings entirely similar to those found in the younger children. There was never more than a trace of urobilinogen present.

The laws governing the formation of the chief bile salts, sodium glycocholate and sodium taurocholate, are for the most part unknown.

Bile Salts and Bile Pigments in Duodenal Juice of Children

Patient	Age	Diagnosis	Day of Experiment	Urobilinogen	Urobilin	Sodium Taurocholate, Gm. per 10 C.c. Duodenal Juice	Sodium Glycocholate, Gm. per 10 C.c. Duodenal Juice	Ratio NaT : NaGly
Os.	2 da.	Icterus neonatorum	1	0	0	....	.....	....
Q.	4 da.	Normal	1	0	0	....	.....	....
Fi.	4 da.	Icterus neonatorum	1	0	0	....	.....	....
Se.	6 da.	Normal	1	0	0	....	.....	....
Fo.	11 da.	Normal	1	0	0	....	.....	....
J.	17 da.		1	0	0	....	.....	....
Ry.	17 da.	Icterus neonatorum	1	0	0	....	.....	....
P.	1 mo.	Normal	1	0	800	23.0	103.7	1:5
			8	0	0			
Cl.	2 mo.	Normal	1	0	800	11.6	83.6	1:7
			8	0	600			
			14	0	0	9.8	158.7	1:15
St.	3 mo.	Icterus neonatorum	1	0	400	....	.....	....
H.	3 mo.	Normal	1	..	...	5.9	12.2	1:2
			5	..	...	9.7		
Sc.	4 mo.	Malnutrition	1	..	...	32.9	241.7	1:7
			21	..	...	75.9	187.3	1:2
			26	..	...	29.9	226.1	1:7
J. W.	4 mo.	Atrophy	1	..	...	21.4	156.7	1:7
			14	..	...	16.8	204.0	1:12
			19	..	...	19.8	116.3	1:6
			35	..	...	**21.1**	**107.4**	**1:3**
			40	**0**	**800**	**39.8**	**163.8**	**1:4**
Ot.	5 mo.	Otitis media	1	..	...	26.7	133.2	1:5
			7	..	...	17.5	48.9	1:3
			28	..	...	**50.6**	**155.3**	**1:3**
			37	..	...	**27.6**	**357.0**	**1:13**
Re.	5 mo.	Atrophy	1	..	...	30.9	125.4	1:6
			28	..	...	**28.3**	**95.2**	**1:3**
T. S.	7 mo.	Rickets	1	..	...	42.1	153.0	1:4
			8	..	...	**14.2**	**108.8**	**1:8**
W.	9 mo.	Normal	1	..	...	14.6	15.3	1:1
G.	9 mo.	Normal	1	Trace	800	27.6	68.0	1:2
			2	Trace	800			
Oh.	10 mo.	Normal	1	0	0	....	.....	....
Ca.	11 mo.	Rickets; congenital syphilis	1	0	600	....	.....	....
			3	0	0			
L.	18 mo.	Malnutrition	1	0	0	....	.....	....
G.	2 yr.	Celiac disease	1	..	...	30.1	520.0	1:17
			10	0	800			
			26	0	800	71.3	119.3	1:2
A.	6 yr.	Celiac disease	1	0	400	30.6	61.8	1:2
			13	0	0	37.9	202.6	1:5

Experimental work on bile fistula in dogs indicates that in health the output of bile salts varies greatly and that the output is greater on a meat than on a carbohydrate diet. It is known that there is a circulation of bile salts. They are reabsorbed from the intestine and reexcreted in the bile. To the pediatrician, they are of particular interest because they lessen intestinal putrefaction, and aid in the absorption of fat and possibly of mineral salts. Sodium taurocholate is supposed to be the more effective in lessening putrefaction.

Jacubowitsch was the first to determine the percentages of taurocholic and glycocholic acids in the bile of infants. He came to the mistaken conclusion that the percentage of taurocholic acid was the greater.[6] Baginsky and Sommerfeld, who next approached the problem, found a decided preponderance of glycocholic acid, the ratios varying from 1.5 : 1 to 13 : 1.[7] Gundobin quotes the work of Orloff and of Geptner. They found that the bile of infants contained about one-half as much bile acid as that of the adult. They also found a preponderance of glycocholic acid, although they thought it less marked than in the adult. Hammarsten's figures for the adult show a varying preponderance for glycocholic acid of from 30 : 1 to 2.25 : 1.

Determinations of bile salts in the duodenal juice have not hitherto been carried out. Specimens were collected after eight hours' fasting. All cloudy specimens containing a mixture of gastric juice were rejected. The bile acids were estimated according to the method of Schmidt and Dart.[8] Twenty-seven specimens of duodenal juice from twelve subjects were analyzed. The youngest infant was 1 month old; the oldest child, 6 years. Five were normal; three had varying grades of malnutrition, one an otitis media, one a mild rickets and two celiac disease. The results are given in the table. Wide variations in the total bile salts were to be expected and a range of from 18 mg. in a healthy infant of 3 months to 550 mg. in a 2-year-old boy with celiac disease was found.

There was a decided preponderance of sodium glycocholate; but this varied from equality to fifteen fold. There is nothing striking in the findings in patients with celiac disease. Patients J. W., Ot., Re., and T. S. were given a course of cod liver oil before the specimens were obtained, the analysis of which are shown in heavy type. No effect could be noted.

SUMMARY

The duodenal juice of infants contains a varying concentration of bile salts of from 0.2 to 5 per cent. There is a fluctuating but decided preponderance of glycocholic acid, and the content of bile salts in the duodenal juice is not influenced by the presence of celiac disease or by the administration of cod liver oil.

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6. Jacubowitsch: Jahr. f. Kinderh. **24**:373-383, 1886.
7. Baginsky and Sommerfeld: Jahr. f. Kinderh. **19**:321-335, 1896.
8. Schmidt and Dart: J. Biol. Chem. **45**:415-421, 1920.

REPORT OF TWO CASES WITH UNUSUAL CALCAREOUS DEPOSITS *

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The two cases here reported present some unusual features. While they differ widely from each other in the fundamental conditions present, they have in common unusual calcareous deposits occurring in the epiphyses of some of the long bones in one patient, and in the skin and subcutaneous tissues in the other.

REPORT OF CASES

Case 1.—A boy, aged 5 weeks, admitted to the Hospital for Sick Children, Nov. 25, 1922, weighing 4 pounds and 11 ounces (2,126 gm.), was markedly emaciated and had a flexion deformity involving almost all of the joints of the body. The arms could not be elevated above the shoulders, the forearms could not be fully extended and the fingers were flexed so that, with the exception of the index fingers, they touched the palm. A slight permanent kyphosis was present. The hips, knees and ankles were also involved so that extension beyond an angle of about 135 degrees was impossible. The feet were everted and flat. All the joints appeared to be enlarged. The lower jaw was very small and receded so much that the upper lip did not approximate the lower one. These deformities are well shown in Figure 1. Examination of the muscles showed secondary contractures. A systolic murmur was heard over the precordial area.

The father and mother, who were first cousins, were quite healthy, and no history could be obtained of any similar condition occurring in any member of either family. The first pregnancy terminated at seven months, the infant being stillborn. The patient, who was the second child, was a full term infant with a birth weight of 6½ pounds (2,954 gm.). The labor was normal, and the deformities as described were present at birth. The baby did not seem able to nurse and took its artificial feeding poorly. After admission, November 25, he gradually became weaker, and died, December 26. The temperature varied from 98 to 102 F. No cause was found for the fever.

Roentgenograms were taken of the wrists, elbows, shoulders, knees and ankles. The following notes from the report of Dr. A. H. Rolph, Department of Radiology, are of interest:

"Peculiar irregular (calcareous?) deposits are present in the epiphyses at both ends of the humeri, the proximal ends of the radii, ulnae and tibiae and the distal ends of the femora. The patellae are practically outlined by these deposits. The distal ends of both radii and ulnae show a narrow zone of rarefaction just proximal to the epiphyseal lines, which is suggestive of congenital syphilis."

Unfortunately, no roentgenograms were taken of the trunk, hips or hands. The deposits were not present in the skin or muscles, or in any situation other than the epiphyses and the patellae. These deposits are shown in Figures 2 and 3.

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* Received for publication, Oct. 26, 1923.

* From the wards and laboratories of the Hospital for Sick Children and the departments of pediatrics of the University of Toronto.

A Wassermann test made with blood obtained from the patient, December 17, was negative; the blood from the mother was also negative. November 25, the red blood count was 4,750,000; hemoglobin, 85 per cent.; white blood count, 13,200, with 65 per cent. polymorphonuclear cells. December 21, the white blood count was 7,800. The intracutaneous tuberculin test was negative. The blood serum calcium, November 30, was 9.3 mg. per hundred cubic centimeters and the inorganic phosphorus, 4.5 mg.

Fig. 1 (Case 1).—Front and side views of infant.

Summary of Postmortem Findings.—No gross abnormalities of the gastrointestinal tract, spleen, liver or lungs were found. The ductus arteriosus was patent, and the wall of the right ventricle of the heart was somewhat hypertrophied. The flexion deformities of the various joints, previously described, were noted. The left patella and right olecranon process were removed for further study.

Fig. 2 (Case 1).—Left arm: Calcareous deposits in the region of the elbow joint.

Fig. 3 (Case 1).—Left leg: Calcareous deposits in the region of the knee joint.

Microscopic examination of the various tissues revealed nothing pathologic in the thymus gland, suprarenals or pancreas. There was slight congestion of the lungs and quite marked congestion of the liver. In both the liver and the spleen were numerous Kupffer cells, showing marked phagocytosis for red blood cells. The kidneys showed marked congestion of the capillaries below the pelvic epithelium and small laminated renal calculi in the pelvis. A few of the renal

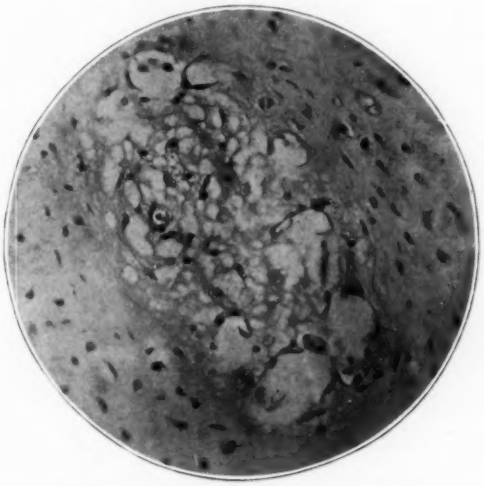

Fig. 4 (Case 1).—Photomicrograph of section of patella: Vacuolated appearance, with areas in which nuclei have disappeared.

Fig. 5 (Case 1).—Photomicrograph of section of patella: Calcareous deposits in degenerated connective tissue.

tubules were dilated and filled with granular débris, in which were calcareous deposits. Examination of the patella showed that the bulk of the section was cartilaginous, but scattered here and there throughout the substance of the cartilage were areas of loose connective tissue This connective tissue was peculiar in that many of the cells appeared to be undergoing degenerative changes,

some of the nuclei being shrunken and the intercellular substance vacuolated and granular. It was in these areas of degeneration that the deposits were found, the cells having entirely disappeared in some places, the space being occupied by rather dense calcareous material. This calcareous material had evidently acted as a foreign body, since an occasional foreign body giant cell was seen at the periphery of these areas. There was no other evidence of any inflammatory reaction (Figures 4 and 5). Somewhat similar areas of degeneration were seen in the connective tissue toward the base of the mitral valve curtains, but here there was no deposition of calcium salts.

CASE 2.—A boy, aged 12 years, admitted to the Hospital for Sick Children, May 17, 1922, was markedly emaciated, with contracture deformities of the knees, hips and elbows. In regard to the skin, itchiness was marked. It had a pinkish tinge and presented a most unusual appearance (Figs. 6 and 7). With the exception of small areas around the umbilicus and on each side of the spinous processes, it was rough, dry and mottled. Scars of old bed sores were present over some of the vertebrae. Other small scars were present, lateral to the sacrum, on the elbows and on the legs. There were some wartlike projections on the right leg and both elbows. These consisted of calcareous material which

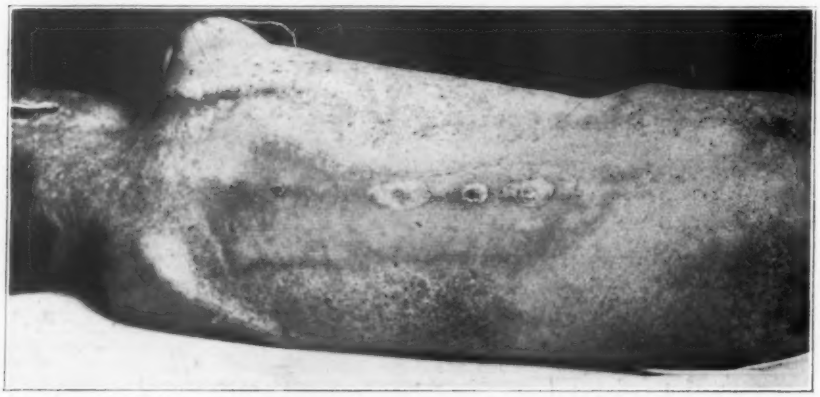

Fig. 6 (Case 2).—Appearance of patient: Unusual skin condition.

appeared to be in the process of erosion through the skin. Some of these were removed quite readily, and areas were left which healed and formed small scars similar to the ones already mentioned. On palpation of the skin of the extremities and certain parts of the trunk, hard plaques of material were felt, varying in size from just palpable spicules to one piece which extended from the right external malleolus up the leg for a distance of about 18 cm., and was from 2 to 4 cm. in width. Examination of the glandular, respiratory, circulatory, digestive and nervous systems disclosed nothing abnormal. The family history was negative.

The boy had apparently been normal until May, 1918, when he complained of pain in his ankles and difficulty in walking. The father noted a small sore on the internal malleolus of one ankle. A few days later, a similar sore appeared on the internal malleolus of the other ankle. He was then brought to the hospital, and attended the outpatient department once a week for three months. At this time, he was able to walk a little, but dragged his legs as though very tired. He was admitted to the hospital in September, and discharged in December, 1918. His condition had become progressively worse, and on discharge he was unable to walk at all. The condition of the skin at that time was apparently as previously described. The contractures of the arms and legs were first noticed

in the spring of 1919, and have gradually become more marked. His mentality has been excellent throughout.

Roentgenograms were taken of the extremities and the trunk. The following report by Dr. A. H. Rolph, Department of Radiology, is of interest:

"Numerous calcium deposits are present in the subcutaneous tissues of both legs and arms. These all appear to be very superficial and not in the muscles as occurs in myositis ossificans. The calcareous material is also widely distributed throughout the subcutaneous tissues of both buttocks." The accompanying roentgenograms show some of these deposits (Figs. 8 and 9).

The Wassermann reaction with the blood was negative. May 17, 1922, the red blood count was 4,150,000; white blood count, 6,200, with 78 per cent. polymorphonuclear cells; the hemoglobin, 75 per cent. The intracutaneous tuberculin test was negative. The blood serum calcium, May 25, was 9.7 mg. per hundred cubic centimeters and the inorganic phosphorus 4.4 mg.

A small nodule was removed from the skin of the right arm above the elbow for microscopic examination. This nodule showed quite normal squamous epithelium and sebaceous and sweat glands. In the subcutaneous areolar tissue was a small calcareous mass surrounded by a rather dense zone of connective tissue.

Fig. 7 (Case 2).—Appearance of patient.

The calcareous material was vacuolated, suggesting the possibility that fatty material had been dissolved out during the process of dehydration in the preparation of the tissue for section. The areolar tissue outside the nodule appeared normal.

COMMENT

In a careful survey of the literature, the authors have been unable to find any case similar to the first reported in this paper. The diagnosis in the second case rests between xeroderma pigmentosum and scleroderma. The following extract is from the notes made on this case by Dr. H. A. Dixon of the sub-department of dermatology:

"Xeroderma pigmentosum (pigmented dry skin) rarely begins after the second year. In the early stages, the exposed parts are involved. The changes are in order, freckle-like pigmentation, white atrophic spots and telangiectasis; later superficial ulceration occurs, and this may be followed by warty growths, malignant epithelioma and a fatal issue.

The ulcers are due to secondary infection and are followed by contractures. The affected skin is especially sensitive to the actinic ray of sunlight and even slight exposure may set up a solar dermatitis.

"Scleroderma (hard skin) when becoming generalized is slowly progressive and may be subject to periods of intermission, months and

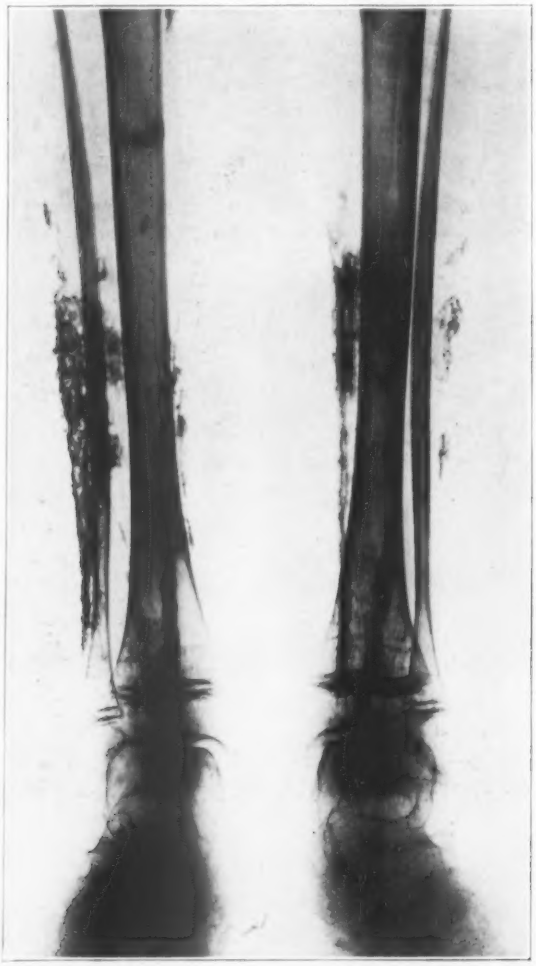

Fig. 8 (Case 2).—Calcareous deposits in the skin and subcutaneous tissues (legs).

even years elapsing before it reaches the final stages. Occasionally after a stationary period, the skin may gradually return to normal. Owing to the atrophy, the subcutaneous fat disappears and the skin becomes bound down to underlying structures. The knees and elbows may become so fixed that flexion is impossible. The vitality of the

skin is so low that slight injury, especially about the prominent parts, such as the elbows, knees and malleoli, may give rise to ulceration and even gangrene. The later phases of the fibrosis may lead to calcareous deposits of calcium phosphate and calcium carbonate.

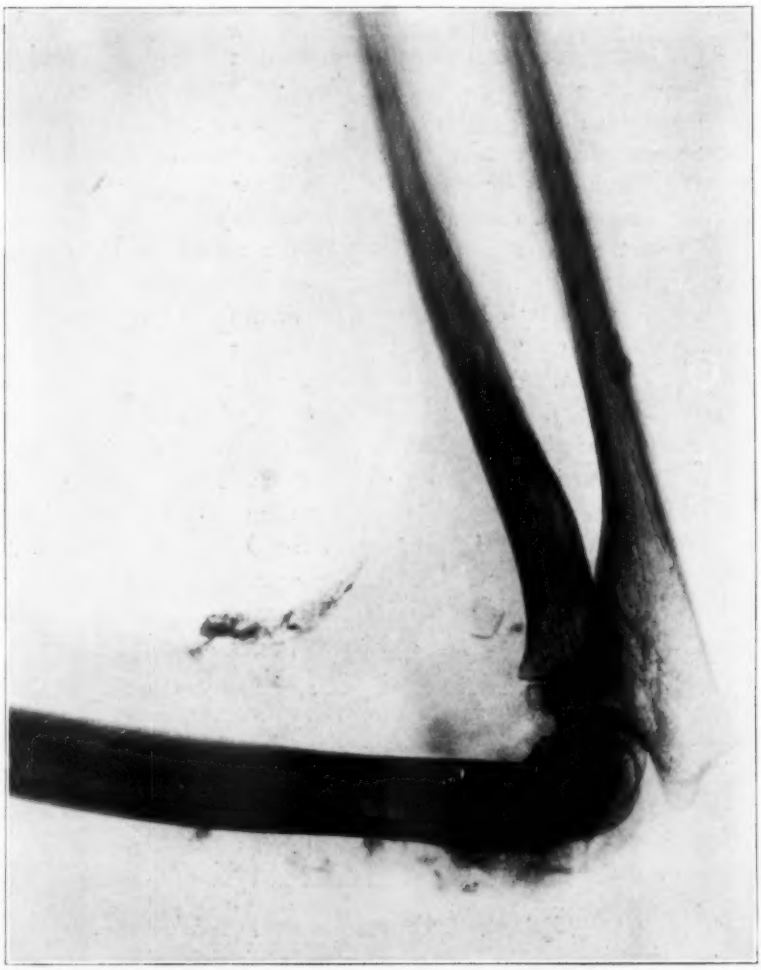

Fig. 9 (Case 2).—Calcareous deposits in the skin and subcutaneous tissues (left arm).

"The present case shows very marked contractures and areas of atrophy and pigmentation. Definite calcareous deposits are also present. In our opinion, this case very closely resembles xeroderma pigmentosum, but in a review of the literature we have been unable to find any records of calcareous deposits in the skin in this disease. On the other

hand, there have been reported a few cases of scleroderma with calcium deposits in the skin, and on this account the weight of evidence is on the side of the latter disease."

The occurrence of calcareous deposits in the skin and subcutaneous tissues has been noted by a number of authors. It is not limited to any one disease. Morse [1] has recently reported an unusual instance of necrosis of the subcutaneous fat in a 3½-year-old girl, which was followed by calcification. Calcareous deposits in the subcutaneous tissue in cases of scleroderma, as already mentioned, have been reported by Hunter [2] and others. Kuznitzky and Melchior [3] have described calcareous deposits in the skin in an atypical case of lipodystrophia progressiva. Under the heading "Ueber Kalkmetastasen in der Haut," Jadassohn [4] reported a case of a 12-year-old boy who had calcareous deposits not only in the skin, but also in the endocardium, lungs and kidneys. This patient had an osteomyelitis of the pelvic bones, and shortly before death staphylococci were obtained from the blood stream. Raynaud's disease may apparently be a factor in the subcutaneous deposition of calcium salts.[2] The presence of calcium in the skin and subcutaneous tissues in a case of leukemia has been noted by Riehl.[5] Also, a number of German authors have reported the occurrence of calcareous deposits in the skin which did not follow any particular disease. These deposits occurred chiefly in the region of the phalangeal joints, along the course of the ulna and at the elbow; and although they are not limited to any one disease, the microscopic findings in the different conditions are almost identical. Inflammatory changes are not a factor in these deposits. In the early stages of calcification, it is unusual to find round cells or giant cells, although in the later stages these calcareous masses may act as foreign bodies and produce some mechanical irritation. The majority of authors have found that the deposition occurs in or around degenerated tissue cells. Jadassohn, however, observed in his case that there was no degeneration of the elastic or connective tissue fibers at the site of some of the recent calcareous deposits. Nevertheless, he considered that probably a toxin had diminished the vitality of these cells, and that this in some way favored the deposition of the calcium salts. Practically all the writers are of the opinion that the deposits do not occur in normal tissue cells.

Under the name of myositis ossificans progressiva, a number of cases have been described with calcareous deposits in the muscles. In

1. Morse, J. L.: Calcification of Skin in a Child, Am. J. Dis. Child. **22**: 412 (Oct.) 1921.
2. Hunter, W. K.: Glasgow M. J. **79**: 241, 1913.
3. Kuznitsky, E., and Melchior, E.: Arch. f. Dermat. u. Syph. **123**: 133, 1916.
4. Jadassohn, J.: Arch. f. Dermat. u. Syph. **100**: 317, 1910.
5. Riehl: Arch. f. Dermat. u. Syph. **117**: 299, 1913.

this condition, the deposits occur not in normal muscle tissue but in a fibrocellular connective tissue which invades the normal muscle and connective tissue cells. This new formed tissue is not the result of any inflammatory change, but is rather a simple hyperplasia of unknown origin.

It is a matter of common experience to find calcareous deposits in various tissues in which there have been degenerative changes. For example, calcareous tubercles in the lung, in the cervical, bronchial or mesenteric glands, are frequently seen. Calcareous plaques in the aorta and other arteries in arteriosclerosis are quite common. Less frequently seen, but still fairly common, are the calcareous deposits in necrotic renal epithelium in some cases of marked nephrosis. Occasionally, one meets with similar deposits in inspissated pus in an old empyema. In fact, one such case recently seen by one of us (I. H. E.) showed a thick layer of calcareous material over the lung at the left apex, at one point of which there was in addition actual bone formation. In all of the above mentioned conditions, the calcification is preceded by degenerative changes, the fatty, necrotic material evidently favoring the deposition of calcium salts.

In the light of these facts, the microscopic appearance of the tissues involved in the cases here reported is of considerable interest. In Case 1, the areas of connective tissue in the patella show numerous vacuoles of various sizes with comparatively few nuclei remaining. This is best illustrated in Figure 5. Figure 6, which is from the same case, shows what appears to be a more advanced stage in the degenerative process, with fairly heavy deposits of calcium in the region of the connective tissue. Quite similar findings are seen in the nodule removed from the skin in Case 2, except that numerous vacuoles are still present in the calcareous areas. It would thus appear that while these two cases present very different clinical pictures, the calcareous deposits in both cases occurred in areas showing previous degenerative changes.

On account of the importance of the concentration of the blood serum calcium and inorganic phosphorus for the deposition of bone in healing rickets [6] and also in the union of fractures,[7] the concentration of these elements was determined in the serum of both cases. The values obtained were within the normal limits, although rather less than the average (average calcium per hundred cubic centimeters of serum, 10.0 mg.; average inorganic phosphorus value per hundred cubic centimeters of serum in children, 5.0 mg).

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6. Howland, John, and Kramer, Benjamin: Calcium and Phosphorus in Serum in Relation to Rickets, Am. J. Dis. Child. **22:** 105 (Aug.) 1921.

7. Tisdall, F. F., and Harris, R. I.: Calcium and Phosphorus Metabolism in Patients with Fractures, J. A. M. A. **79:** 884 (Sept. 9) 1922.

The only previously recorded determinations of the calcium concentration in the blood of a patient with subcutaneous deposits of calcium are those reported by Mosbacher,[8] in 1918. He placed his patient on a diet with a low calcium content, then on a diet with a high calcium content. On the first diet, he obtained a value of 18 mg. calcium per hundred cubic centimeters of blood; and on the latter, a value of 43 mg. However, his determinations of the blood calcium in a normal individual under the same conditions were 17 and 28 mg. of calcium per hundred cubic centimeters, respectively. In view of the known constancy of the calcium concentration in blood serum of normal individuals, it is obvious that no importance can be placed on these determinations.

The results of our investigations throw no further light on the exact cause of the deposition of the calcareous material. However, from our present knowledge, it appears that it is the local condition and not a general metabolic change which is the important factor determining the deposition of calcium salts in the unusual cases here recorded.

SUMMARY

Two unusual cases with calcareous deposits have been reported. In the first case, the deposits occurred in the epiphyses of most of the long bones. The patient lived only a few weeks, and had a peculiar deformity of the jaw and limitation of the movements of the other joints of the body. We were unable to find a report of any similar case in the literature.

The calcium deposits in the second case were in the skin and subcutaneous tissues. The underlying condition appeared to be either scleroderma or xeroderma pigmentosum.

Blood serum calcium and inorganic phosphorus determinations were made in both cases. The values found were 9.3 mg. and 9.7 mg. of calcium per hundred cubic centimeters; and 4.5 and 4.4 mg. of phosphorus. These determinations are essentially within the normal limits.

From the microscopic examination of the tissues and the determination of the blood serum calcium and inorganic phosphorus, it would appear that the local condition is the important factor which determined the deposition of the calcium salts in these cases.

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8. Mosbacher, E.: Deutch. Arch. f. klin. Med. **128**: 107, 1918.

TREATMENT OF EARLY HEREDITARY SYPHILIS WITH INTRAMUSCULAR INJECTIONS OF SULPHARSPHENAMIN *

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The peculiar difficulties of administering any form of intravenous therapy to small infants are well known. The median longitudinal sinus has proved most valuable as a route for giving fluids intravenously, or for transfusion, but, occasionally, attempts to administer arsphenamin and neo-arsphenamin intrasinusly have been attended with fatal results. At times, the superficial veins of the scalp are prominent and may be utilized, but often the external jugulars are the only veins available, and at times even these are not visible. It is often difficult, except for those who have had much practice, to make a clean puncture into a jugular vein which is covered by skin, fascia, and platysma muscle. As a result, various attempts have been made to prepare arsenicals in a form suitable for subcutaneous, intramuscular and rectal administration. Neo-arsphenamin, specially neutralized for intramuscular injection, can now be obtained, but its use is attended by the frequent formation of indurated areas, and occasionally sloughs have occurred. It is not the purpose of this paper to discuss the relative merits of these different forms of antisyphilitic therapy, but to record the results obtained by intramuscular injections of sulpharsphenamin in the treatment of young infants suffering from hereditary syphilis. The sulpharsphenamin reported on is sulfarsénol (the French brand). We are now making tests with the American made drug in a series of cases and expect to publish the results shortly.

Sulpharsphenamin was first described in September, 1919, by Levy-Bing, Lehnhoff-Wyld and Gerbay.[1] In chemical structure, it is closely related to neo-arsphenamin, its molecular formula ($C_{12}H_{11}As_2N_2CH_2OSO_2Na$) differing from the latter only by the addition of a single oxygen atom. It is a pale yellow powder, having, in solution, an odor

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* From the Harriet Lane Home, Johns Hopkins Hospital, and the Department of Pediatrics, Johns Hopkins University.

* This clinical trial of sulpharsphenamin was suggested by Dr. Carl Voegtlin of the United States Public Health Service, who furnished us with a supply of the drug for our studies.

1. Levy-Bing, Lehnhoff-Wyld, and Gerbay: Un nouveau composé arsenical: Le sulfarsénol, Ann. d. mal. ven. **14**:520 (Sept.) 1919.

faintly suggestive of carbon bisulphid. The above mentioned authors demonstrated that it was more stable than arsphenamin or neo-arsphenamin (there was practically no change in composition after standing twenty-four hours in test tubes exposed to the air); that it was from one-half to one-third as toxic for mice as neo-arsphenamin, and that it was more soluble than either arsphenamin or neo-arsphenamin. They obtained good results in a series of thirty-three adults, whom they treated for a short time.

Since this first publication, the drug has been given a rather wide clinical trial intravenously (Levy-Bing, Lehnhoff-Wyld and Gerbay;[1] Bernard,[2] Gallonnier[3]), subcutaneously (Emery and Morin;[4] Mendes Da Costa;[5] Yernaux and Bernard[6]), and intramuscularly (Galonnier;[3] Mendes Da Costa;[5] Chatellier;[7] Duroeux;[8] Levy-Bing and Gerbay[9]) by a number of European clinicians. Many of them are enthusiastic about their results. However, Papegaaij and Rinsema[10] did not consider sulpharsphenamin given subcutaneously as effective as neo-arsphenamin given intravenously, either in the rapidity of the disappearance of spirochetes from mucous lesions, or in the frequency with which a negative Wassermann reaction could be obtained. None of the clinical trials, so far, have dealt with infants.

Voegtlin, Johnson, and Dyer[11] have aroused new interest in this drug by their studies of the synthesis, and chemical and pharmacologic properties. This preparation is now being manufactured by several American firms.

DOSAGE

The arsenic content of neo-arsphenamin is 19 per cent., while that of sulpharsphenamin is 21 per cent. The dose of neo-arsphenamin usually

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2. Bernard, R.: Le sulfarsénol, Scapel **19,** October, 1919.

3. Galonnier, P.: Recherches sur l'administration et l'elimination de sulfarsénol au cours de la syphilis, Toulouse, 1920.

4. Emery and Morin, A.: Traitement de la syphilis par les injections sous-cutanees d'arsénobenzènes, Bull. méd. **34**:549 (June) 1920.

5. Mendes Da Costa, S.: Sulfarsénol als antisyphiliticum, Nederl. Tijdschr. v. Geneesk. **2**:513 (Aug. 7) 1920.

6. Yernaux and Bernard: Contribution au traitement de la syphilis par le sulfarsénol, Scapel **24**:593 (Dec.) 1919.

7. Chatellier, L.: Le traitement de la syphilis et de quelques dermatoses par le sulfarsénol intramusculaire, Toulouse méd. **5**:176 (March) 1920.

8. Duroeux, L.: De l'influence du sulfarsénol sur la réaction de Bordet-Wassermann dans le traitement de la syphilis, Progrès méd. **36**:155 (April 9) 1921.

9. Levy-Bing and Gerbay: Essai d'une therapeutique arsenicale intramusculaire, Ann. d. mal. ven. **15**:15 (Jan.) 1920.

10. Papegaaij, J., and Rinsema, P. G.: Mededeelingen over Sulfarsénol, Nederl. Tijdschr. v. Geneesk. **2**:560 (July 30) 1921.

11. Voegtlin, C.; Johnson, J. M., and Dyer, H.: Sulpharsphenamine: Its Manufacture and Chemotherapeutic Properties, Pub. Health Rep. **37**:2783-2798 (Nov. 10) 1922.

given is 15 mg. per kilogram of body weight. Because of the approximate similarity in the content of arsenic, most clinicians have taken this figure as the maximum dose for sulpharsphenamin. One manufacturer of sulpharsphenamin states that when arsenic is well tolerated, the dose may be increased until 15 mg. per kilogram of body weight is given. Duroeux,[8] in treating adults, began with small doses (0.12 gm.), increasing the dosage up to 0.60 gm. In testing the human tolerance for the drug, Levy-Bing, Lehnhoff-Wyld and Gerbay [9] gave a dose as large as 0.9 gm. to an adult without provoking any reaction. In determining the toxic dose for mice, they found that the toxicity of sulpharsphenamin was from one half to one third that of neo-arsphenamin. Voegtlin, Johnson, and Dyer,[11] in working with sulpharsphenamin, demonstrated that the minimum lethal dose for albino rats was from 320 to 480 mg. per kilogram. The official U. S. regulations governing the sale of neo-arsphenamin required only that it pass a test of 200 mg. per kilogram. Experimental investigation of the power of the different arsenicals to penetrate the tissues has shown that the larger the dose the greater the penetration.[12] For this reason; in view of the lower toxicity, and because it is well known that babies usually will tolerate proportionately larger doses of active drugs than adults, it seemed to us that the dose might be increased. Accordingly, after observing that the smaller doses were well borne, we began to use larger amounts, and are now giving doses of 20 mg. per kilogram of body weight. We have given this dose many times, but have never seen a serious reaction.[13] This means that an infant receives 1.47 times as much arsenic as we previously would have given him in the form of neo-arsphenamin.

The twenty-one cases on whom our report is based were seen in the course of routine examinations in the Harriet Lane Home and Dispensary. All of the patients were less than 1 year of age, and presented the clinical picture of early hereditary syphilis. The diagnosis in all but one case was confirmed by Wassermann tests of the blood. In one patient, in whom the suspicion of syphilis was strong because of a positive Wassermann reaction in the mother and the presence of snuffles, treatment with arsenic was delayed until the age of 3 months, when his Wassermann reaction had become positive. In the interim, treatment

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12. Voegtlin, C.; Smith, H. W.; Dyer, H., and Thompson, J. W.: Penetration of Arsenic into the Cerebrospinal Fluid, etc., Pub. Health Rep. **38**: 1003-1013 (May 11) 1923.

13. In a number of cases the dose was increased to 22 or 23 mg. per kilogram. In all but one case, this dose was tolerated. In this case, the injection was followed by a rather severe gastro-intestinal disturbance. As the incident occurred in the month of July, when such disturbances are relatively common in otherwise normal infants, we did not feel certain that it was a result of the arsenical treatment.

with mercury by inunctions was administered. In a number of the patients, diagnosis was aided by the characteristic roentgenographic picture of the extremities. So far as possible, a record was kept of the individual lesions—snuffles, skin rashes, condylomas, rhagades, pseudoparalyses, etc.—and their duration after the beginning of treatment was noted. The Wassermann test was not made after each treatment but usually only at the beginning of each new course.

METHOD

Treatments were given at weekly intervals until a course of six injections had been completed. The patients were then allowed a rest period of two months before a second course was commenced. During the rest period, mercury inunctions were given. A portion of one-third strength blue ointment (diluted mercurial ointment, U. S. P.) about the size of a pea was applied each morning on a flannel abdominal binder. The exertions of the infant (crying, kicking, etc.) furnished the friction necessary for absorption. Our plan of treatment is to give three complete courses of sulpharsphenamin after a negative Wassermann reaction has been obtained. Four complete courses will be, then, the minimum number that any child will receive.

At the time of this publication, all of our twenty-one patients have completed one course; seven, two courses, and four, three courses.

Sulpharsphenamin has been given by others, intravenously, subcutaneously and intramuscularly. We have used the intramuscular route entirely, most of the injections being made into the muscles of the buttocks. It has seemed to us that fewer local reactions were obtained when precautions were taken to insure that all of the solution went into the muscles and none into the subcutaneous tissue. A sharply pointed needle of fine bore (No. 22) is used with a 2-c.c. Luer syringe. The solution to be injected is usually less than 1 c.c. in volume. This is drawn into the syringe, and then the rest of the syringe is filled with air. The site selected for the injection is cleaned with iodin and alcohol and the needle is wiped with dry sterile gauze. With the syringe held in a perpendicular position, the needle is plunged directly into the muscle tissue. The injection can then be made so that the solution enters the muscle first, and the last drops can be ejected from the needle by forcing the air in the syringe into the muscle. The needle is withdrawn slowly to allow the tissues time to contract and prevent return of the solution along the needle tract.

COMMENT

It is generally agreed that there are fewer and less intense local reactions from the preparation when concentrated solutions occupying a small volume are used. We have been obtaining the drug in ampules

containing 0.6 gm. The contents of one of these is dissolved in 2.7 c.c. of sterile, freshly distilled water. The total volume will then be 3.0 c.c., a 20 per cent. solution. The maximum dose is reached when the baby is given 0.1 c.c. of this solution per kilogram of weight.

General reactions following the intramuscular injection of sulpharsphenamin are not common, and when present are mild. We have on several occasions noted slight gastro-intestinal disturbances (vomiting and increase in the number of stools) of about twenty-four hour's duration. These reactions have never been so severe or so frequent as those following the intravenous injection of neo-arsphenamin. Doble,[14] in contrast to the experience of the French investigators, found that, in a few of his patients, a Herxheimer reaction, which was very slight and transitory, occurred. With one of our patients (J. P.), a white male infant, 2 months of age, who had been having convulsions, and who had spinal fluid pleocytosis, the number of cells in the spinal fluid increased after his first and second treatments. However, there was no noticeable constitutional reaction, and the infant gained 15 ounces (425 gm.) during the period. After the third treatment, the convulsions ceased.

Levy-Bing and Gerbay [9] found that in the majority of their patients there was no local reaction. Some of them complained of a sensation of heaviness and muscular stiffness, which rarely lasted more than from twenty-four to forty-eight hours. Emery and Morin [4] had a similar experience. Papegaaij and Rinsema,[10] who had given about 1,100 intramuscular injections in the buttocks, found that there was acute pain of short duration following all injections; that induration at the site of injection was uncommon, and that abscesses never occurred. Our experience with infants is somewhat similar. Pain is more difficult to recognize in an infant than in an adult. However, we have not received complaints from the mothers of dispensary patients, nor have the babies under our own observation in the hospital, exhibited signs of pain, except at the time of the injection. Up to the present time, we have made 349 injections. In twenty-four instances, or 7.7 per cent. of the cases, an indurated area from 1 to 2 cm. in diameter developed by the following week. There was never any evidence that these "lumps" were tender or painful, but they disappeared rather slowly (in from one to three months). We have never seen an abscess or a slough develop following the intramuscular injection of sulpharsphenamin.

In our series, there were four babies who had the characteristic maculopapular eruption of congenital syphilis. In one of these, the eruption disappeared seventeen days after the beginning of treatment.

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14. Doble, F. C.: Sulfarsénol in the Treatment of Syphilis, Lancet **2**:243, 1920.

In the other three patients, it was absent one week after the second injection had been given. In all four of these patients, improvement commenced two or three days after the first treatment. The rash appeared less elevated and the color had begun to fade. There were four colored infants who had circinate syphilids of the face and extremities. In all of these, the skin was practically clear when they were brought back for the second treatment, except for slight pigmentation, which rapidly became less evident after subsequent treatments.

Two infants had anal condylomas. Improvement was noticed during the first week, but healing was not complete until the end of the third week in one patient and the fourth week in the other. Rhagades and fissures of the lips were present in two patients. In one of these, healing occurred after two treatments; in the other, only after the completion of a course of six treatments.

Difficult and noisy respiration due to swelling and discharge from the nasal mucous membranes ("snuffles") is one of the most constant findings in early hereditary syphilis. This symptom was present in sixteen of our twenty-one patients. In only one of the infants was the nasal discharge bloody. The administration of arsenic nearly always results in improvement in this symptom, but since there is actual hypertrophy of the mucous membranes, and at times underlying periostitis, its final disappearance must be slow. After the first course of treatments, the "snuffles" had entirely disappeared in four patients, and was improved in nine and unchanged in one. In the two remaining cases, the notes on the duration of the symptom were not complete.

There were five infants who had pseudoparalysis. In each of these patients, pain, tenderness and swelling had subsided markedly when they were brought back for the second treatment. In one of them, the movements were entirely normal after the first treatment. Three required two treatments and one three treatments before the symptom completely disappeared. In each of these patients, the nature of the lesion was confirmed by the characteristic roentgenographic picture.

Our notes in regard to the decrease in size of enlarged spleens and epitrochlear glands are not sufficiently complete to enable us to draw any conclusions regarding the result of treatment.

Despite the limitations of a purely laboratory method of diagnosis, the Wassermann test is and probably will remain the most important aid in the diagnosis of syphilis and in determining the effect of treatment. Duroeux,[8] in a series of 162 adults treated with weekly injections of sulpharsphenamin, noted that the Wassermann reaction became negative in 68 per cent. after one course (ten injections), in 19 per cent. after two courses, and in the remaining 13 per cent. after three courses. He was able to obtain even better results by giving the injections at more frequent intervals. The Wassermann tests in our series were

made by the syphilis department of the Johns Hopkins Hospital according to the technic described by Keidel and Moore.[15] Of the twenty patients whose Wassermann reaction of the blood were positive, eleven, or 55 per cent., gave negative reactions after the completion of one course. Since, in most instances, the second Wassermann test was not made until the beginning of the next course of treatments, this means that the reaction had become negative and remained so over a period of two months without arsenic treatment. In one patient, a negative reaction was obtained one week after the first injection, and this negative reaction was permanent. One patient with a positive reaction after one course gave a negative reaction after two courses. In the one patient whose reaction remained positive after two courses, very small doses had been used. After a third course, in which the size of the dose was increased, the reaction became negative.

Throughout the period of treatment, it was repeatedly noted that the general condition of the patients was markedly improved; that they seemed less susceptible to infections, gained in weight, and gradually regained the appearance of normal infants.

Two illustrative case histories are given:

REPORT OF CASES

CASE 1.—*History.*—W. B., boy, white, aged 5 weeks, admitted to Harriet Lane Home, Oct. 31, 1922, with a diagnosis of early hereditary syphilis, gave the following history: The father, aged 27 years, had been treated for syphilis, and the mother, aged 29 years, had a positive Wassermann reaction. The patient was the second child. The first was born prematurely (at eight months) and died in two days. The birth of the patient was normal. The present complaint was: (1) eruption over the skin, of five days' duration, and (2) snuffles, of three weeks' duration.

Physical Examination.—The patient was fairly well developed and well nourished. There was a red, slightly elevated, maculopapular eruption over the face, with some incrustation and a marked fissuring of the lips, extending into surrounding skin for one quarter inch (0.6 cm.). Over the extremities, there was an eruption of the same nature, which was symmetrically distributed. There were many maculopapular lesions varying, in size from one-quarter to three-eighths of an inch (0.6 to 0.9 cm.) in diameter. Many of these were confluent. There was a slight scaling on the wrists and some desquamation on the palms. The inguinal glands were palpable; the epitrochlears were not enlarged. There was a yellowish discharge from each nostril. No distension or rigidity of the abdomen was noted. There was a slight umbilical hernia. The spleen was 2 cm. below the left costal margin. The remainder of physical examination was negative.

Laboratory Examination.—The white blood count was 7,500; the urine was negative. Culture of the nasal discharge was negative for *B. diphtheriae*. The blood Wassermann reaction was positive. A roentgenogram of the extremities revealed evidences of syphilitic osteochondritis (Fig. 1).

Treatment.—Administration of sulpharsphenamin was begun, Nov. 7, 1922, in the routine manner, except that two treatments each week were given in

15. Keidel, A., and Moore, J. E.: Wassermann Reaction in the Johns Hopkins Hospital, Bull. Johns Hopkins Hosp. **34**:16 (Jan.) 1923.

the first course until eight treatments had been given. Following the first injection, the eruption faded rapidly and by the seventeenth day (Nov. 24), after four injections, it had entirely disappeared (Fig. 2).

This course of treatment was completed, December 5, and the patient was discharged for six weeks. The mother was instructed to apply diluted mercurial ointment by means of an abdominal binder daily. The patient had improved greatly and was discharged weighing 10 pounds and 13½ ounces (4,832.6 gm.). Jan. 30, 1923, the blood Wassermann reaction was negative.

The second course, six treatments, was started, January 30. There was no reaction, either local or general, to any of the injections. The patient now,

Fig. 1 (Case 1).—Appearance of patient before treatment was commenced, showing maculopapular eruption.

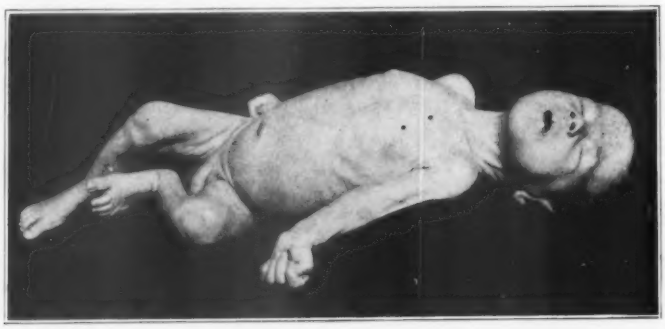

Fig. 2 (Case 1).—Appearance of patient after seventeen days' treatment (four injections), showing condition of skin.

except for slight scars from old rhagades and a palpable spleen, showed no signs of hereditary syphilis. The Wassermann reaction was negative; he was gaining in weight, and was normal in appearance for his age. May 15, the blood Wassermann reaction was negative.

The third series, six treatments, was started, May 8. There was only one slight local reaction from treatment, in form of a lump in the buttocks. The patient has now received three courses of treatments. The Wassermann reaction has been negative since the first course. There are no signs of hereditary syphilis except the scars about the lips and a palpable spleen. The patient is 9 months old, weighs 20 pounds and 12 ounces (9.43 kg.) and in appearance is a normal healthy child.

CASE 2.—*History and Examination.*—E. B., girl, colored, aged 6 weeks, admitted to Harriet Lane Dispensary, May 5, 1923, with a diagnosis of early hereditary syphilis, on admission had a definite pseudoparalysis of both upper extremities, and cried when she was handled. There was a maculopapular eruption on the extremities and macular lesions on the palms and soles. The epitrochlears and the spleen were palpable. The blood Wassermann reaction was positive. A roentgen-ray examination of the extremities revealed evidences of syphilitic osteochondritis.

Treatment.—One course of six weekly injections of sulpharsphenamin was given. The only reaction was a small lump in the right buttock after one injection. After the second treatment, the eruption disappeared, and the movements of the upper extremities were normal. The Wassermann reaction was negative two months after one course of treatment.

CONCLUSIONS

1. Sulpharsphenamin is a safe and easily administered arsenical preparation for the treatment of hereditary syphilis in infants.

2. Sulpharsphenamin can be safely given to infants in a dose as large as 20 mg. per kilogram of body weight.

3. The reactions, either general or local, to treatment are very slight.

4. In 55 per cent. of the patients, the Wassermann reaction became negative after one course of six treatments.

5. The symptoms of hereditary syphilis rapidly disappear, and the patient's general condition quickly improves after treatment.

6. Because of the ease with which sulpharsphenamin can be administered, it should be very useful to the practitioner who is not adept at intravenous therapy.

SUMMARY OF TWENTY-ONE CASES

C. R.—Snuffles, slight improvement after 1 course of treatment; circinate syphilid, disappeared after 2 treatments; palpable spleen, remained palpable after 1 course of treatment; positive blood Wassermann reaction, negative after 1 course of treatment.

M. W.—Snuffles, slight improvement after 1 course of treatment; enlarged spleen, no change after 1 course of treatment; epitrochlears enlarged, no change after 1 course of treatment; anal condylomas, improved rapidly and were healed 1 week after 4th weekly injection; positive blood Wassermann, negative after 1 course of treatment.

B. R.—Enlarged spleen, no change after 1 course of treatment; enlarged epitrochlears, no change after 1 course of treatment; anal condylomas, completely healed after 3 treatments; positive blood Wassermann reaction, remained positive after 1 course of treatment.

L. H.—Snuffles, no note made; cracked lips, and rhagades, sores on lips healed rapidly after 2d treatment; macular syphilid, completely healed after 2 treatments; enlarged spleen, no change; negative blood Wassermann reaction, remained consistently negative during 2 courses of treatment.

E. C.—Pseudoparalysis, right arm, child moved arm normally after 2 treatments; positive blood Wassermann reaction, was positive after 1 course, doubtful positive after 2 courses of treatment and negative after 3 courses.

C. B.—Bloody nasal discharge, improved after 1 course of treatment; circinate syphilid, disappeared after 2 treatments; enlarged spleen, no change

after 1 course of treatment; enlarged epitrochlears, no note made; positive blood Wassermann reaction, negative after 1 course of treatment.

A. H.—Snuffles, disappeared after 4 treatments; pseudoparalysis of both legs, moved normally after 1 treatment; enlarged epitrochlears, no change after 1 course of treatment; positive blood Wassermann reaction, remained positive after 1 course of treatment.

E. B.—Snuffles, markedly improved after 4 treatments; maculopapular syphilid, disappeared after 2 treatments; pseudoparalysis (arms), movements of arms normal after 3 treatments; enlarged spleen, no improvement after 1 course of treatment; positive blood Wassermann reaction, negative after 1 course of treatment.

H. H.—Snuffles, disappeared after 4 treatments; enlarged spleen, no note made; positive blood Wassermann reaction, negative after 1 course of treatment.

J. McK.—Snuffles, improved after 1 course of treatment; positive blood Wassermann reaction, remained positive after 1 course of treatment.

L. F.—Positive blood Wassermann reaction, remained positive after 1 course of treatment.

W. B.—Snuffles, greatly improved after 1 course of 8 treatments; maculopapular syphilid, had disappeared by 17th day after treatment was stared; rhagades and cracked lips, healed by end of 6th treatment; enlarged spleen, no note made; positive blood Wassermann reaction, negative after 1st course of 8 treatments and has remained negative for 7 months with 2 consecutive courses of 6 treatments each.

J. B.—Snuffles, disappeared after 1st course of treatment; pseudoparalysis (both legs), movements normal after 1 treatment; positive blood Wassermann reaction, negative after 1 course of treatment and has remained so for 7 months with 2 consecutive courses of treatment.

J. C.—Positive blood Wassermann reaction, negative after 1 course of 4 treatments, and has remained so for 4 months with 1 subsequent course of treatment.

R. P.—Snuffles, greatly improved after 3 treatments; pseudoparalysis, upper and lower extremities, movements normal after 2 treatments; positive blood Wassermann reaction, remained positive after 4 treatments.

I. G.—Snuffles, greatly improved after 1 course of treatment; positive blood Wassermann reaction, negative after a single injection.

O. P.—Snuffles, no improvement after 1 course of treatment; positive blood Wassermann reaction, negative after 1 course of treatment.

I. F.—Snuffles, no note made; maculopapular syphilid, healed by end of 2nd treatment; palpable spleen, no improvement after 1 course of treatment; positive blood Wassermann reaction, remained positive after 1 course of treatment.

J. S.—Circinate syphilid, only slight pigmentation of skin remained after 2 treatments; positive blood Wassermann reaction, remained positive after one course of treatment; negative after two courses.

E. W.—Snuffles, improved rapidly as soon as treatment was begun and had almost entirely disappeared by end of 1st course of treatment; rhagades, greatly improved and had healed by end of 1st course of treatment; positive blood Wassermann reaction, negative after 1st course of treatment and has remained negative for 5 months with one subsequent course of treatment.

H. W.—Snuffles, slight improvement after 1 course of treatment; positive blood Wassermann reaction, Wassermann was "suggestive negative" after 2 treatments and negative after 5 treatments.

VITAL CAPACITY AS AN AID IN DIAGNOSIS OF TRACHEOBRONCHIAL ADENOPATHY IN CHILDREN FROM FIVE TO TWELVE YEARS OF AGE

ITS SIGNIFICANCE IN EVALUATION OF VITAL CAPACITY MEASUREMENT OF AVERAGE NORMAL CHILDREN *

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In a previous paper on the measurement of vital capacity of children, we indicated that the low values obtained by this test in some of our apparently normal subjects was probably due to tracheobronchial adenopathy.[1] The evidence in support of this view was obtained from the roentgenologic findings, in all those instances studied by this method at the time that the work was prepared for publication. The interpretation of a relationship existing between the vital capacity of the lungs and tracheobronchial adenopathy is one of considerable importance, since it implies (1) that the measurement of the vital capacity can be used as a valuable adjunct in the diagnosis of tracheobronchial adenopathy and (2) that, in the attempt to establish a standard of vital capacity for the average normal child, the incidence of this disease would materially modify the result.

At the outset, it is essential to summarize the present conception of the diagnosis, incidence and clinical significance of tracheobronchial adenopathy in children from 5 to 12 years of age, as indicated by a survey of the recent literature.

The diagnosis is considered one of great difficulty, as the physical signs are indefinite. D'Espine's sign is most generally used.[2] Its value, however, is considered uncertain, because of the discrepancy in the findings and interpretations of various investigators.[3] The roentgen-ray interpretation of hilum, root and trunk shadows is also attended by

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1. Wilson, May, and Edwards, D. J.: Standards for Normal Vital Capacity for Children; the Lung Capacity in Certain Intrathoracic Conditions, Am. J. Dis. Child. **22**:443-454 (Nov.) 1921.

2. Much, H.: Tuberculosis of Children (translated by Dr. Marx Rothschild), New York, MacMillan Company, 1921, p. 93.

3. Griffith, J. P. C.: Diseases of Infants and Children, Vol. 2, Philadelphia, W. B. Saunders Co., 1919, p. 556.

some uncertainty, differences in opinion being due to the variation in technic as well as to the fact that the norm for children is difficult to standardize.[4] There seems to be a general agreement that an increase in the hilum shadow is indicative of engorgement and enlargement of the intrathoracic glands, and that the accompanying extensive linear markings radiating to the lung periphery, particularly the upper parts of the lung, may be considered pathologic.[5]

As to the clinical significance of tracheobronchial adenopathy, two important facts are stressed:[6] 1. Engorgement of the intrathoracic glands accompanies and is a sequel of most infections, particularly those involving the respiratory tract, such as measles and pertussis. 2. The seat of tuberculous infection, both latent and active in children, is in the intrathoracic glands.[7] These considerations suggest that the incidence of tracheobronchial adenopathy in any group of children is likely to be high, and that the degree and permanency of the involvement will vary with the etiologic factor.

Although realizing the difficulties of any comparative study based on still uncertain criteria, as is evident from the summary of the diagnosis as outlined above, we purpose in this investigation to make a comparative study of (1) the vital capacity and roentgen-ray findings of an arbitrarily selected group of average children, apparently normal and ranging in age from 5 to 12 years, and (2) the vital capacity and roentgen-ray findings of a selected group of children with tracheobronchial adenopathy.

The subjects selected for comparative roentgenographic study and vital capacity measurement may be considered under two general groups. The first group comprises eighty-three subjects, arbitrarily selected from a total of 360 apparently normal children (who were observed in the investigation for the normal standards of vital capacity [1]). All of these children passed a routine physical examination. They were apparently healthy, and were attending public or private school in New York City. We shall subdivide this group into two divisions, one made up of fifty average white children selected without regard to race, and the other thirty-three colored children. The other main group of subjects considered in this study were selected because of predisposing history, clinical symptoms, physical signs or roentgen-ray evidence justifying the clinical diagnosis of tracheobronchial adenopathy.

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4. Pottenger, F. M.: Clinical Tuberculosis, St. Louis, C. V. Mosby Company, 1922, p. 114.

5. Dunham, Kennon: Stereoroentgenographic Studies of Pulmonary Tuberculosis, Troy, N. Y., Southard Company, 1915, p. 522.

6. Fleischner, E. C.: Bronchial Lymphadenopathy, Nontuberculous, J. A. M. A. **79**:175 (July 15) 1922.

7. Fishberg, Maurice: Pulmonary Tuberculosis, Ed. 2, Philadelphia, Lea and Febiger, 1919, p. 395.

METHOD AND TECHNIC

The vital capacity measurement was determined as described in the preliminary investigations.[1] All known factors which tend to diminish the vital capacity measurement were considered, and all subjects giving evidence of such complicating factors were eliminated from this study.

A graph of the vital capacity readings of 360 average, apparently normal children, is included for comparative study (Fig. 7).

Fig. 1.—Normal subject with a grading of 1 +.

The roentgenologic examinations, with films, were supplemented by confirmatory fluoroscopic and stereoscopic examinations in the majority of cases. The roentgen-ray technic used, in the main, was that described by Dunham and Blackfan.[8] In a small number of cases, serial films were taken over a period of a year or more. The degree and the extent of the hilum shadow and the pulmonary markings were arbi-

8. Dunham, H. K., and Blackfan, Kenneth: Preliminary Report on Clinical and X-Ray Findings in the Chests of Normal Children, Tr. Nat. Tuberculosis Ass'n, 1921.

trarily graded; those indicating little evidence of increase in the hilum shadow with lung parenchyma fairly free and clear (Fig. 1) as 1 +; those showing definite increase in the hilum shadow with pulmonary markings extending midway to the lung periphery (Fig. 2) as 2 +, and those revealing extensive increase in the hilum shadow with linear markings pronounced and extensive radiating to apices and lung periphery (Fig. 3) as 3 +.[9]

Since the completion of this investigation, there has been published by the National Tuberculosis Association, a report containing an excel-

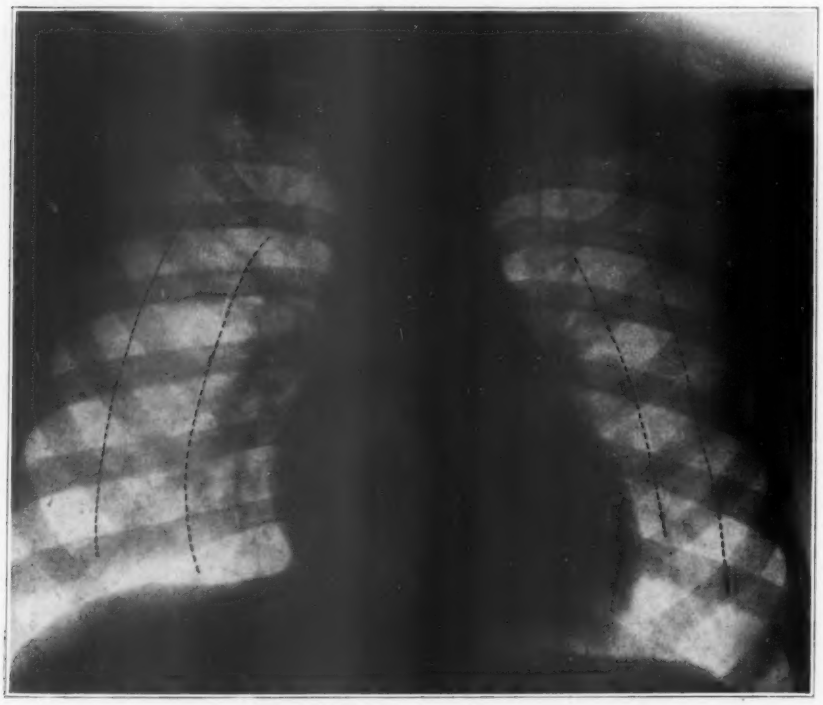

Fig. 2.—Normal subject with a grading of 2+.

lent composite diagrammatic roentgenogram of the normal chest of the child, showing a division of the lung into three vertical zones, for "convenience of description" (Fig. 5). The arbitrary divisions which we have made in our preliminary and present reports, i. e., the gradations of 1 +, 2 + and 3 +, are comparable to the division of Zone I, Zone II, and Zone III, respectively, shown in their illustration.

9. These notations were made without the knowledge of vital capacity readings, and were checked three different times by us. Likewise, many of the films were seen, and our interpretations corroborated by disinterested, competent observers.

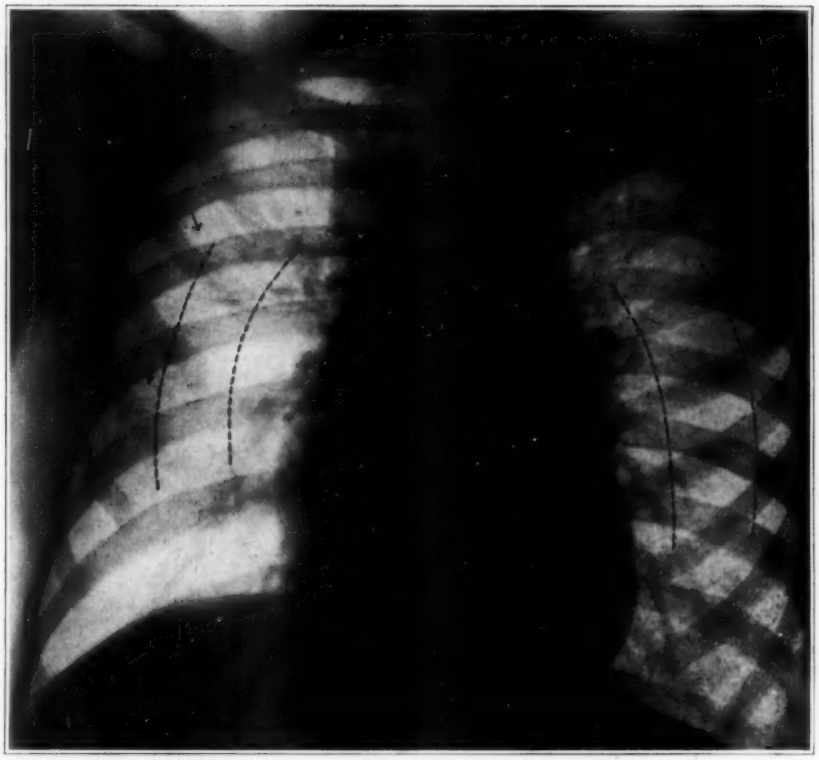

Fig. 3.—Normal subject with a grading of 3 +.

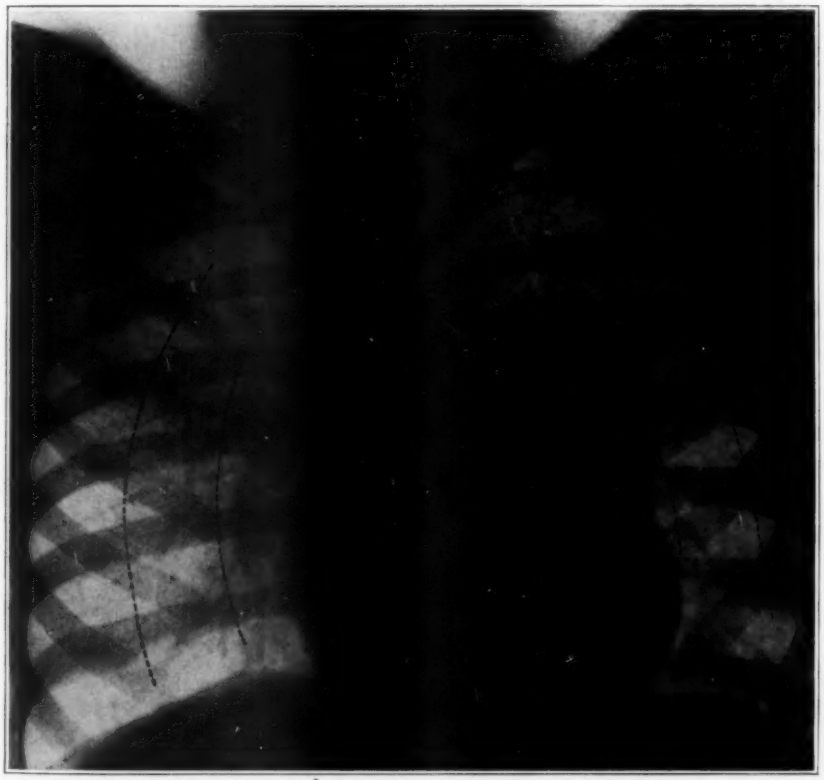

Fig. 4.—Chest of subject with tracheobronchial tuberculosis.

RESULTS

A comparative analysis of the roentgen-ray findings and vital capacity values observed for the normal group, as shown by Table 1, A and B, indicates that 16 per cent. of the white subjects and 84 per cent. of the colored subjects, with roentgen-ray findings graded as

Fig. 5.—Composite diagrammatic roentgenogram of the normal chest of the child, showing the three zones (reproduced by courtesy of the American Review of Tuberculosis).

3 +, showed average reductions in the vital capacity of 17 and 22 per cent., respectively, for each group. The remaining normal subjects, with findings graded as 2 + and 1 +, had vital capacity values within the average normal range, with slightly higher values for the 1 + group.

TABLE 1.—*Vital Capacity Readings and Roentgen-Ray Findings of Normal Subjects from 5 to 12 Years of Age*

	Roentgen Ray Graded 1+; Vital Capacity, Liters per Square Meters of Surface Area	Roentgen Ray Graded 2+; Vital Capacity, Liters per Square Meters of Surface Area	Roentgen Ray Graded 3+; Vital Capacity, Liters per Square Meters of Surface Area
A—White Subjects:	1.74 2.00	1.57 1.85 1.93	1.52
	1.85 2.04	1.72 1.81 1.92	1.52
	1.80 2.00	1.76 1.85 2.08	1.68
	1.81 2.15	1.72 1.80 2.00	1.66
	1.86 2.16	1.77 1.81 2.23	1.64
	1.81 2.19	1.75 1.81 2.28	1.65
	1.97 2.20	1.76 1.90 2.31	1.70
	1.90 2.20	1.75 1.97 2.45	1.60
	2.07	Average, 1.91	Average, 1.62 (a reduction of 17%)
	Average, 1.98		
B—Colored Subjects:	1.80	1.77	1.36 1.47 1.61
	2.04	1.74	1.33 1.46 1.66
	Average, 1.92	1.74	1.38 1.46 1.66
		1.85	1.48 1.43 1.65
		2.09	1.42 1.55 1.68
		Average, 1.83	1.40 1.51 1.60
			1.47 1.54 1.66
			1.40 1.51 1.74
			1.48 1.51
			Average, 1.51 (a reduction of 23%)

TABLE 2.—*Roentgen-Ray Findings and Vital Capacity Readings of Thirty Subjects from 5 to 12 Years of Age, with Tracheobronchial Adenopathy*

Type of Case	Vital Capacity, Liters	Roentgen-Ray Findings
Tuberculous:		
Active	1.12 (−42%)	All graded 3+
	1.35 (−32%)	
	1.42	
	1.47	
	1.42	
	1.43	
Latent	1.58 (−18%)	
	1.50	
	1.54	
	1.64	
	1.70	
Nontuberculous:		
Recent infection	1.41 (−28%)	
	1.43	
	1.48	
	1.44	
	1.44	
	1.53	
	1.59	
	1.58	
	1.60	
	1.74	
Asthma group	1.42	
	1.46	
	1.47	
	1.45	
	1.33	
	1.50	
	1.65	
	1.54	Graded 2+
	1.80	Graded 2+

The individual vital capacity values are, in the main, consistent with the average values obtained. These observations are in accord with those found in the preliminary investigation.

The result of the comparative analysis of the vital capacity readings and roentgen-ray findings in our second group of subjects (those with tracheobronchial adenopathy) is shown in Table 2. The roentgen-ray findings in all but two of the subjects of the asthma group are graded 3+, confirming the clinical diagnosis. An average reduction of vital capacity of 23 per cent. was obtained for the group. There were greater individual reductions noted, particularly in subjects with symptoms of active infection, as much as 42 per cent. in one instance. These results

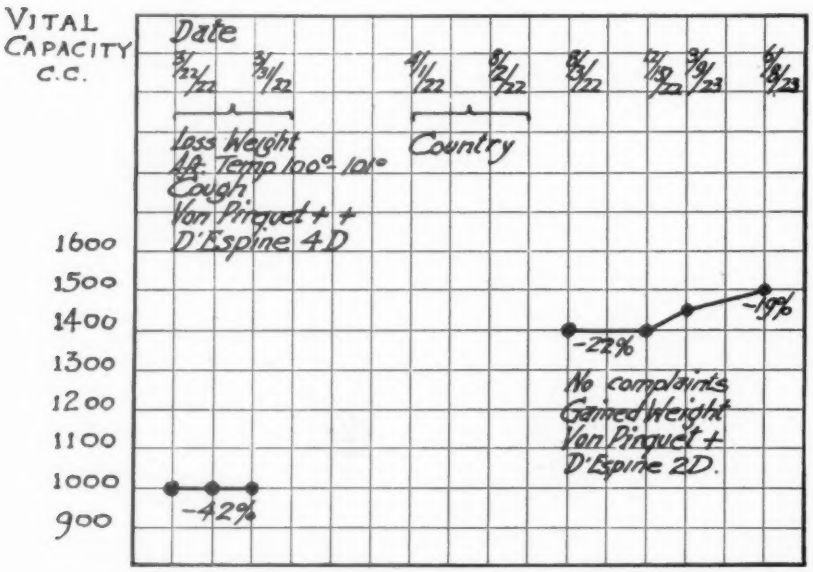

Fig. 6.—Record of vital capacity of girl (J. I., colored), 8 years of age, with tracheobronchial tuberculosis.

show a close correlation between the vital capacity, roentgen-ray findings and clinical diagnosis.

Some of the group with tracheobronchial adenopathy have been observed for a period of one or two years. The record of subject J. I. with tracheobronchial tuberculosis (Fig. 6) is of interest. It shows a reduction of vital capacity of 42 per cent. during the stage of active clinical symptoms. At periods of six months and one year later, when the condition was quiescent, the vital capacity still showed a reduction of 22 and 19 per cent., respectively. During the latter period, the child was apparently well and was attending school. Certain subjects with nontuberculous tracheobronchial adenopathy seemed to show a more

rapid increase in the vital capacity than tuberculous subjects, a few coming within normal limits within from three to six months.

A comparative analysis of the plotted curves of combined vital capacity readings for subjects graded as 1 +, 2 + and 3 +, and the curve of vital capacity readings of 360 average normal children (Fig. 6) is of interest. It shows rather strikingly that subjects with a grading of 1 + and 2+, respectively, fall within the normal range, 1.74 and 2.14 liters per square meter of body surface, while subjects with a

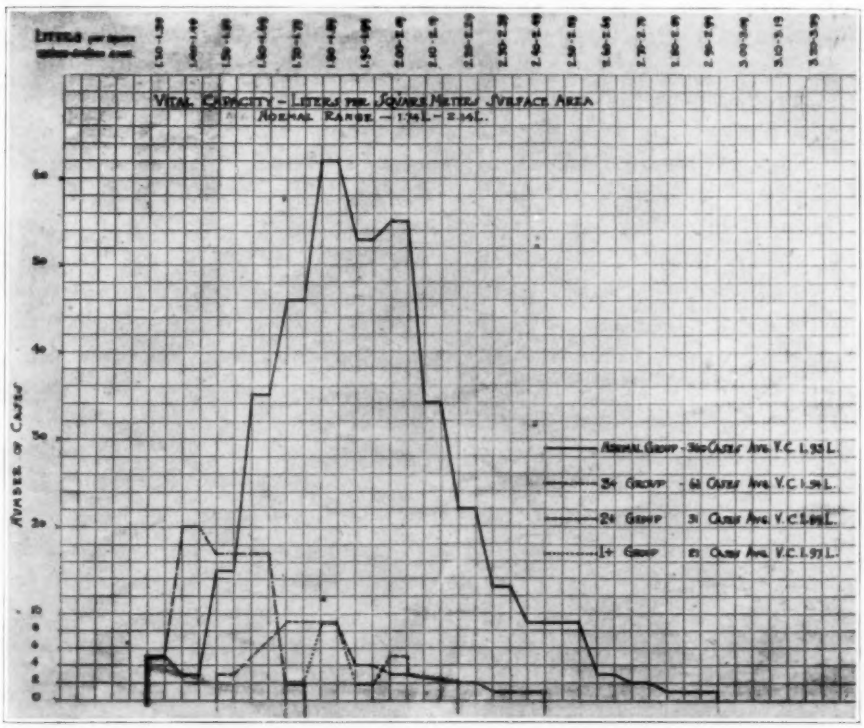

Fig. 7.—Comparative vital capacity curves of 360 average normal subjects, and subjects with tracheobronchial adenopathy.

grading of 3 + cover the lower range of the normal curve, from 1.30 to 1.69 liters per unit of body surface.

COMMENT

We have attempted to contrast the vital capacity readings and the roentgen-ray findings obtained from a study of average children, apparently normal, with similar observations on subjects with clinical symptoms or physical signs of tracheobronchial adenopathy. We will first consider the results of our observations on normal subjects.

The most significant feature of these results is the roentgen-ray findings observed in 16 per cent. of normal white subjects and in 84 per cent. of colored subjects. The roentgen-ray findings described in these apparently normal children, which we have graded as 3 +, may be considered as pathologic. An explanation of the failure to eliminate these subjects from the normal group by anamnesis and physical examination may be found in the unreliability of a history, and the recognized difficulty of eliciting diagnostic physical signs of tracheobronchial adenopathy, as well as the frequent absence of these signs. Of clinical significance is the reduction of vital capacity obtained for the subjects graded as 3 +, averaging 17 per cent. for the white subjects and 23 per cent. for the colored subjects. These findings indicate that, among a group of apparently normal children, one may expect to find a certain percentage of subjects who will show a diminished vital capacity, and will give accompanying roentgenologic evidence of tracheobronchial adenopathy. This would seem to be particularly true of colored subjects, possibly because of their susceptibility to such infections as tuberculosis.

A further consideration of the roentgen-ray findings in normal subjects giving vital capacity readings within normal limits lends additional support to the conclusion that the shadows observed, and graded as 1 + and 2 +, respectively, are to be interpreted as within the normal range. This view coincides with the findings reported by the National Tuberculosis Association, previously referred to.

Of clinical significance is the close relation between the roentgen-ray findings and the vital capacity readings of these subjects. This is well brought out by a comparison of the superimposed curves of combined vital capacity readings (Fig. 7). They show that subjects with a grading of 3 + cover the lower range of the normal curve, indicating that these individual low vital capacity readings may be due to tracheobronchial adenopathy. The fact that subjects with a grading of 1 + and 2 + fall within the normal range indicates, we believe, that the roentgen-ray findings have no bearing on the vital capacity measurements. These considerations make it possible to predict with reasonable confidence that apparently normal children revealing a vital capacity reading within normal limits will present, by roentgen ray, a normal picture according to currently accepted standards; and that children exhibiting a diminished vital capacity will show (other factors known to diminish the vital capacity being excluded) roentgenographic evidences of tracheobronchial adenopathy.

Our observations of the group with clinical symptoms and physical signs of tracheobronchial adenopathy are of interest in that they show a close relation between roentgen-ray findings, vital capacity and clinical diagnosis. The vital capacity measurement is of clinical significance,

therefore, in that it affords corroborative evidence concerning the clinical course and the diagnosis. Of interest are the low individual vital capacity values observed in the group with symptoms of active infection. Though the number observed is too few for any final conclusions, these observations tend to indicate that a reduction of vital capacity in subjects with tracheobronchial adenopathy of more than 25 per cent. is suggestive of an active infection; while a reduction of vital capacity in similar subjects of from 15 to 25 per cent. is suggestive of a healed or latent infection. While in no way may the diminished vital capacity observed indicate the etiologic factor, these observations support the view that a reduction of vital capacity of more than 15 per cent. supplies corroborative diagnostic evidence of tracheobronchial adenopathy.

Since at the present time the pathology underlying the roentgen-ray findings observed is inconclusive, any explanation as to the diminished vital capacity observed is speculative. However, if we consider the shadows observed as caused by connective tissue changes, due to either tuberculous or nontuberculous infection, healed or active, it is reasonable to assume a loss of lung elasticity, which may be expected to reduce the vital capacity measurement. To substantiate this assumption, extensive observations controlled by careful necropsy would be necessary. Such a study is not possible in children, but it has seemed practical in animals, and we are undertaking such an investigation.

SUMMARY

1. Diminished vital capacity and roentgenologic evidence of tracheobronchial adenopathy were found in 16 per cent. of apparently normal white subjects, and 84 per cent. of apparently normal colored subjects.

2. A reduction of vital capacity of more than 15 per cent. in an apparently normal child indicates the necessity of a roentgenologic examination.

3. The possible presence of tracheobronchial adenopathy must be considered in the evaluation of the vital capacity measurement in apparently normal children.

4. A reduction of vital capacity of from 15 to 42 per cent. was found in children with tracheobronchial adenopathy.

5. The vital capacity is an aid in the diagnosis of tracheobronchial adenopathy.

CONGENITAL DEFECT OF THE SKIN OF THE NEW-BORN *

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Congenital defect of the skin constitutes one of the rare and interesting anomalies peculiar to the new-born infant. Recently, a striking example of the condition came under our observation, and in view of the vast extent of the skin defect present in this infant, together with the rarity of its occurrence, a report of the case seems desirable.

From the cases recorded in the literature, it is apparent that the lesions most frequently involve the scalp. They do occur, however, on the trunk and extremities, and in these locations the skin defects are often bilateral and also may be fairly symmetrical in their extent on the two sides of the body. The defects are present in early fetal life; thus they undoubtedly originate in utero. The margins of the involved areas are sharply defined and slightly elevated. Microscopic examination shows a failure in development, not only of the epidermis, but also of the glands, smooth muscle and adipose tissue of the skin. The presence of extensive defects of the skin is not incompatible with the life of the infant, and with proper treatment, areas of wide extent may heal spontaneously and without resort to skin grafts.

Various theories have been advanced to explain the cause of this interesting condition, but none are entirely satisfactory and convincing. In view of the uncertainty as to the etiology, we will content ourselves with a detailed report of the case.

REPORT OF CASE

Mrs. B., the mother of the baby having the extensive skin defect, was 31, and had been married five years. She was the mother of two healthy normal children. With her third and last pregnancy, the last menstrual period began July 28, and ended July 31, 1921. Life was first felt about November 7, 1921, and the expected date of confinement was April 8, 1922. On September 18, 1921, the mother underwent cholecystectomy, following which she promptly developed a marked jaundice associated with pale colored stools. After the operation, she had bearing down pains, with slight blood spotting, which lasted a few days.

The patient remained in a rather poor physical condition with low blood pressure, slight fever, rapid pulse, slight leukocytosis and continued jaundice, with bilirubin in the urine.

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On account of her poor general condition, coupled with the persistent jaundice, labor was induced, Feb. 27, 1922, with the hope of securing a viable child, and also of alleviating the mother's condition by terminating the pregnancy. The course during the puerperium was fairly normal. There was some improvement in her general health and diminution in the jaundice, but the stools remained clay colored.

At birth, the baby weighed 1,610 gm. The placenta was small, round, friable and bright red, with a considerable area of degeneration present. The defect of the skin in this infant extended as a band 1.5 cm. wide across the anterior part of the abdomen above the umbilicus, and on the lateral sides of the trunk spread upward over the ribs and down to the crest of the ileum. The photograph (Fig. 1), taken March 8, 1922, or nine days after the birth of the infant, shows the extent and distribution of the skin defect on the left side of the body. A small area present on the lateral aspect of the arm, is not shown. At birth, the area was covered by a thin, delicate, translucent membrane, through which fine blood vessels, and also the ribs, were distinctly visible. Apparently, the muscles in these regions were also absent to some extent. Small skin defects about 2 by 3.5 cm. in size were also present on the lateral aspects of each thigh and arm. Linear scars running upward along the posterior axillary fold, and downward over the crest of the ileum connected these small areas with the large one on each side of the trunk, suggesting that at one time the skin defects may have been larger than at birth, and that some healing had occurred in utero. The symmetry of the extent and of the distribution of the lesions on the two sides of the body was striking.

Two days after birth, the thin membrane had broken down, and the area was covered by an opaque yellowish crust, from beneath which there was a slight oozing of serum. A few days later, the epithelium at the margins of the area became reddened and slightly thickened, and slowly began to replace the crust covering the area of the skin defect. The healing process steadily progressed in this manner, and about seventeen days after birth, the areas on the thighs and arms were completely covered by epithelium. When the baby was 2 months old, the large areas on each side of the trunk were also practically healed, except for a small region on each side about 1.5 cm. in diameter. Subsequently, the healing process progressed to completion, leaving slight scarring and retraction of the skin in the involved areas, as shown in Figure 2.

At first, the treatment consisted in dusting the area with a powder of equal parts of calomel and bismuth, over which sterile gauze dressing were applied. Because of a tendency for the gauze dressings to adhere to the crusts covering the lesion, we impregnated the gauze dressings with paraffin. This procedure obviated the difficulty we had encountered, and proved most satisfactory. With this treatment, the lesions healed completely and spontaneously, and without resort to skin grafts. The child is still living and in good health.

Instead of entering into a discussion of the literature dealing with this subject, we refer the readers to the complete bibliography published by Abt [1] in 1917. We wish, however, to point out the fact that the distribution of the skin defect in the case reported by Hahn [2] in 1841 greatly resembled that of the skin defect in our case.

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1. Abt, I. A.: Congenital Skin Defects, Am. J. Dis. Child. **14**:113 (Aug.) 1917.

2. Hahn: Angeborener Defekt eines grossen Theils der Haut des Rumpfes, J. d. Chir. u. Augenh. **30**:156, 1841.

Fig. 1.—Appearance of child on the ninth day after birth, showing the extent and distribution of the skin defect on the left side of the body. A small area present on the lateral aspect of the arm is not shown.

Fig. 2.—Appearance of baby when about 5 months of age, showing the complete healing of the area of skin defect.

SUMMARY

In the case of a new-born infant, which at birth had an extensive congenital defect of the skin, the symmetry of the lesions on the two sides of the body was striking.

The most satisfactory treatment consisted in dusting the region with powder composed of equal parts of calomel and bismuth, over which gauze dressings impregnated with paraffin were applied. With this treatment, complete healing occurred without resort to skin grafting.

AMINO-ACID CONTENT OF BLOOD OF INFANTS AND CHILDREN *

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Preliminary to beginning some studies on the amino-acid metabolism of infants and children, it was deemed advisable to obtain figures that would establish with reasonable accuracy the normal fasting amino-acid content of the blood. Owing to the comparatively short period of time that has elapsed since the development and introduction of the Folin method [1] of amino-acid determination, the literature, to date, contains no such observations for use as normal standards.

In the accompanying tables are submitted the results of studies made by the Folin method in fifty cases selected to range from infancy to puberty.

To carry on these observations, it was necessary to choose subjects already in the hospital. While these, of course, are hospital cases they are hospital normals so far as their selection can be controlled. Only patients whose convalescence was definitely established, in whom no kidney impairment existed and whose temperatures were normal were observed. They had been receiving for several days the usual diet for their age. The blood was obtained in the morning, with the subject fasting since the evening meal of the preceding day. In the case of infants, feedings were withheld for six hours.

Tables 1 and 2 give the findings in infants and children.

It has been found for infants and children that there is a variation from 3.92 to 7.14 mg. in 100 c.c. of normal fasting blood; with an average figure of 5.42 mg. per hundred cubic centimeters of blood. The fairly uniform values obtained throughout the periods of infancy and childhood show also that weight does not affect amino-acid values.

In addition to the amino-acid, determinations of the nonprotein nitrogen,[2] of the uric acid [3] and, on as many as possible, of the creatinin [2]

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* Received for publication, Sept. 27, 1923.

* From the Children's Medical Service Massachusetts General Hospital.

1. Folin, D., and Wu, H.: System of Blood Analysis: New Colorimetric Method for Determination of Amino-Acid Nitrogen in Blood, J. Biol. Chem. **51**:377 (April) 1922.

2. Folin, D., and Wu, H.: System of Blood Analysis: Methods for Analysis of N.P.N. and Creatinine, J. Biol. Chem. **38**:81 (May) 1919.

3. Folin, D.: System of Blood Analysis: Revision of the Uric Acid Method, J. Biol. Chem. **54**:153 (Sept.) 1922.

TABLE 1.—*Determinations of Blood Amino-Acid Nitrogen* of Normal Infants*

Case	Age	Sex†	Amino-Acid	N. P. N.	Uric Acid	Creatinin	Diagnosis
1	9 da.	♂	6.02	30.3	2.80	1.13	G. C. conjunctivitis
2	14 da.	♀	5.04	20.1	1.60	....	Staph. conjunctivitis
3	16 da.	♂	6.93	20.4	3.32	....	Staph. conjunctivitis
4	21 da.	♂♀	6.65	19.8	2.32	....	?G. C. conjunctivitis
5	21 da.	♂	4.83	30.0	3.20	1.17	G. C. conjunctivitis
6	4 wk.	♂	5.95	19.2	4.44	....	G. C. conjunctivitis
7	6 wk.	♂	5.25	28.8	3.08	1.23	G. C. conjunctivitis
8	6 wk.	♂♀	5.39	30.6	2.56	0.87	G. C. conjunctivitis
9	8 wk.	♂♀	5.25	25.5	1.28	....	G. C. conjunctivitis
10	10 wk.	♀	5.53	30.3	....	....	G. C. conjunctivitis
11	11 wk.	♂	6.37	15.0	1.44	....	G. C. conjunctivitis
12	3 mo.	♂	4.41	19.8	1.50	0.96	Pyloric stenosis
13	5 mo.	♂	4.76	21.7	2.04	0.96	Bronchopneumonia
14	7 mo.	♂	5.67	25.2	1.80	1.81	Regulation feeding
15	7 mo.	♂	6.02	41.4	6.04	....	Fracture of femur
16	11 mo.	♀	5.95	26.4	3.16	1.28	Bronchopneumonia
17	13 mo.	♂	6.23	27.9	2.48	....	Cleft palate
18	20 mo.	♀	4.90	28.5	2.48	....	Lacerated lid o.d.
19	22 mo.	♂	4.90	37.5	6.76	....	Phlyctenular keratitis
20	22 mo.	♀	5.32	33.0	6.60	....	Phlyctenular keratitis
Maximum			6.93	41.4	6.76		
Minimum			4.41	15.0	1.28		
Average			5.57	26.6	3.10		

* Milligrams per hundred cubic centimeters of blood.
† In this column, ♀ indicates female, ♂ male.

TABLE 2.—*Determinations of blood Amino-Acid Nitrogen of Normal Children*

Case	Age, Years	Sex	Amino-Acid	N. P. N.	Uric Acid	Creatinin	Diagnosis
21	2	♂	4.48	28.8	4.84	....	Rickets
22	3	♂♂	4.69	27.6	4.04	....	Congenital syphilis
23	3	♂♂	4.90	30.9	3.20	1.08	Prolapse of rectum
24	4	♂♂	4.83	22.8	3.48	....	Impetiginous keratitis
25	4	♀	5.04	30.6	3.04	....	Otitis media
26	4	♀	5.46	25.8	2.84	....	Mastoiditis
27	4	♂♂	5.04	32.1	5.16	....	Acute arthritis
28	4	♂♂	5.32	29.1	3.28	1.09	Mastoiditis
29	4	♂♂	5.67	26.7	2.88	....	Postaural abscess
30	5	♂♂	5.74	49.5	5.88	1.29	Esophagoscopy
31	5	♂♂	5.88	26.7	4.24	....	Chronic endocarditis
32	5	♀	5.53	27.9	4.16	....	Mastoiditis
33	7	♂♀	5.74	25.5	4.16	....	Pleural effusion
34	8	♀♀	4.76	30.3	3.16	1.05	Brachial cyst
35	8	♀	6.09	32.4	4.64	1.18	Phlyctenular keratitis
36	8	♂♂	5.56	24.0	3.32	....	Mastoiditis
37	8	♂♂	3.92	27.3	2.56	1.19	Fractured femur
38	9	♂♂	4.90	28.2	....	....	Mastoiditis
39	9	♂♂	5.60	25.4	4.00	....	Mastoiditis
40	10	♂♂	5.18	25.4	4.80	....	Mastoiditis
41	11	♂♂	5.67	31.2	2.92	....	Chorea
42	11	♂♂	4.69	30.3	5.08	....	Contracted orbit o.d.
43	11	♂♂	4.48	24.9	3.96	....	Traumatic eye
44	12	♂♂	5.18	27.9	3.20	....	Hypopyon ulcer o.s.
45	12	♂♀	4.90	27.3	4.40	....	Phlyctenular keratitis
46	12	♀	5.74	25.1	2.96	....	Hair lip; cleft palate
47	13	♂	7.14	....	3.60	....	Endocarditis
48	13	♂♂	4.69	24.6	3.24	....	Fractured femur
49	14	♂♂	5.74	22.7	3.56	....	Mastoiditis
50	15	♂	5.95	28.2	....	....	Mastoiditis
Maximum			7.14	49.5	5.88		
Minimum			3.92	22.7	2.56		
Average			5.28	28.2	3.80		

were made for sake of comparison and as a further check on the normalcy of the group.

In Table 3 are given the findings in a series of normal adults (physicians and research assistants in the Massachusetts General Hospital). These determinations were made for the sake of comparing results obtained in these adults with the same technic and solutions as were used for the infants and children, also for comparison with results reported in adults by Professor Folin [4] and a larger unpublished series by Cobb, Lennox and O'Connor.

In Folin's series of twelve young men, he found a maximum value of 7.8 mg. and a minimum of 5.7 mg., with an average of 6.4 mg.; a slightly lower figure than that obtained by us.

The figures of Cobb, Lennox and O'Connor correspond more closely to those given in Table 3.

TABLE 3.—*Determinations of Blood Amino-Acid Nitrogen of Normal Adults*

Case	Age, Years	Sex	Amino-Acid	N. P. N.	Uric Acid	Creatinin
1	30	♂	8.12	33.6	4.68	1.40
2	24	♀	6.79	29.7	4.16	1.32
3	29	♂	7.56	30.3	4.28	1.47
4	27	♀	6.36	27.6	3.68	1.40
5	31	♂	7.35	28.8	3.72	1.28
6	25	♀	6.51	27.6	3.28	....
7	27	♂	6.94	29.8	4.75	1.53
8	24	♂	7.28	33.3	3.60	1.26
9	28	♂	6.44	34.2	4.28	1.34
Maximum			8.12	34.2	4.75	1.53
Minimum			6.36	27.6	3.28	1.26
Average			7.04	30.5	4.05	1.37

The figures presented show that while age plays no great part in the amino-acid blood content in the group here considered, during infancy and childhood the average amount present is lower than that for adults.

A number of observations obtained in children with definite organic disease showed distinct deviations from the normal range.

4. Folin, D., and Berglund, H.: Retention and Distribution of Amino-Acids with Special Reference to Urea, J. Biol. Chem. **51**:395 (April) 1922.

REVIEW OF THE LITERATURE FOR 1921 AND 1922 ON NEUROLOGY OF CHILDREN *

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MENINGES

A number of new symptoms, some of them observed by Brudzinski in irritative and inflammatory processes of the meninges, have been described by Jonscher;[1] for instance, flexion of the contralateral extremity following pressure on the upper arm or thigh, and a contralateral reflex on flexion of one arm. They are referred to hyperalgesia and, as the well known neck phenomenon of Brudzinski, are genetically brought in relation with the altered state of body colloids.

Meningitis in infancy is rather infrequent, especially under 3 months of age; and then it is caused by certain micro-organisms that rarely play a rôle later. J. V. Cooke and H. H. Bell,[2] in reviewing the literature of the last five years, encountered only twelve cases of meningococcus meningitis in infants under 3 months of age. In a series of 218 cases of tuberculous meningitis in children under 3 years of age, only three infants, and among 104 children with influenzal meningitis only ten, were under 3 months old. Cases of streptococcus, staphylococcus and pneumococcus meningitis are more frequent. Apparently peculiar to the very young are infections with colon bacilli and allied intestinal bacteria; aerogenes bacteria, *B. mucosus-capsulatus, B. pyocyaneous* and *B. typhosus.* The authors add their own observations of meningitis in early infancy due to a bacillus belonging to the paracolon group and due to the bacterium elasticum, hitherto undescribed. Greenthal[3] observed purulent meningitis, caused by *Bacillus acidi-lactici,* in an infant 1 month old, and Sherman[4] observed this condition in a new-born infant. Smith and Aberd[5] isolated the bacterium enteritidis in a case, and Miller and Lyon,[6] a threadlike diphtheroid micro-organism.

Influenzal Meningitis.—Influenzal meningitis is obviously a disease of infancy. This becomes evident from various statistics relative to the incidence of the affection. The study of 692 cases of purulent

* Received for publication, Oct. 6, 1923.

1. Jonscher, K.: Pedjatrja pol **2**, 1922, abstr. Jahrb. f. Kinderh. **49**:175, 1922; and Monatschr. f. Kinderh. **24**:377, 1922.

2. Cooke, J. V., and Bell, H. H.: Incidence of Meningitis in Early Infancy, Am. J. Dis. Child. **24**:387 (Nov.) 1922.

3. Greenthal, R. M.: Case of Meningitis Due to Bacillus Acid-Lactic, Am. J. Dis. Child. **21**:203 (Feb.) 1921.

4. Sherman: Tr. Am. Pediat. Soc. **34**:192, 1922.

5. Smith, John, and Aberd, M. B.: Lancet **2**:705 (Oct.) 1921.

6. Miller, M. K., and Lyon, M. W., Jr.: Am. J. M. Sc. **162**:593 (Oct.) 1921.

meningitis carried out by the department of health of New York City during the past ten years and published by Josephine B. Neal [7] revealed that, among the thirty-two influenza patients, only two were more than 5 years old and sixteen were children of 1 year or less. T. M. Rivers,[8] who reviews 197 cases of various writers and reports twenty-three new cases, found that 79 per cent. of cases occurred in children under 2 years of age. The mortality among 220 patients was 92 per cent. He remarks that no clinical signs were peculiar to influenzal meningitis, so that, especially in infants, undoubtedly the disease escaped recognition, having been mostly mistaken for pneumonia. Smears of the spinal fluid practically always showed gram-negative pleomorphic bacilli. Blindness, paralysis and deafness were the usual sequels. In the majority of cases, the meningitis was possibly a primary disease, produced by a strain of influenza bacilli closely allied to each other but differing from the ordinary respiratory strains. At times, it was secondary, and was caused by the ordinary respiratory strains.

An instance of influenzal meningitis (without necropsy) has been closely studied by Abt and Tumpeer.[9]

Meningococcus Meningitis.—Whenever the fluid obtained by lumbar puncture is clear in suspected meningococcus meningitis, injection of 5 to 10 c.c. of antimeningococcus serum is recommended by Sainton and Schulmann.[10] This is said to be liable to transform the clear fluid into the usual purulent fluid within a few hours by inducing an influx of leukocytes and meningococci.

Among twelve children, all under 3 years, with epidemic meningitis, observed by Squarti,[11] the mortality was 58 per cent. The author emphasizes that the disease may occur in fulminating form so that the sudden death may have medicolegal importance, and that there exists also a pseudotyphoid, a walking and an ephemeral masked form with variable evanescent symptoms. In one of the four atypical cases, a child was convalescent from typhoid when the meningitis developed, and both the typhoid bacillus and the meningococcus were isolated in the spinal fluid.

Root [12] reports the second case known to literature of meningococcus meningitis at the age of 5 weeks, probably acquired at birth, with obstructive hydrocephalus during the acute stage.

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7. Neal, Josephine B.: Arch. Pediat. **38**:1 (Jan.) 1921.
8. Rivers, T. M.: Influenzal Meningitis, Am. J. Dis. Child. **24**:102 (Aug.) 1922.
9. Abt, I. A., and Tumpeer, I. H.: Influenzal Meningitis, Am. J. Dis. Child. **21**:444 (May) 1921.
10. Sainton, P., and Schulmann, E.: Bull. méd., Paris **35**:465 (June) 1921; abstr. J. A. M. A. **77**:323 (July 23) 1921.
11. Squarti, G.: Pediatria **30**:198 (March) 1922; abstr. J. A. M. A. **78**:1426 (May 6) 1922.
12. Root, J. H.: Meningococcus Meningitis with Obstructive Hydrocephalus in the Newly-Born, Am. J. Dis. Child. **21**:500 (May) 1921.

Lewkowicz [13] offers evidences that meningococcus infection takes place through the choroid plexus, so that the inflammatory process develops first in the ventricles and spreads from here to the subarachnoid spaces. Woringer's [14] two cases support this assumption. The logical treatment in case of block would be intraventricular injection of specific serum. Howell and Cohen [15] recommend intraventricular, and in certain conditions, subdural treatment in meningococcus meningitis. One of the factors responsible for the still too high mortality rate and incidence of sequels, even with careful intraspinal injections, is the failure of the serum to reach the foci of inflammation because of the basilar arachnoid spaces being shut off from those above, because of the closure of the small formina of Magendi and Luschka by inflammation or by sagging, the inflammation of the ventricles and the formation of pockets over the cortex of the brain. The serum, therefore, must be introduced above the block and flow over the affected areas. Howell and Cohen, who since 1914 have given early intraventricular injections of serum to infants, have obtained encouraging results. In some cases, the introduction of serum subdurally at the outer edge of the fontanel has yielded marvelous results, and evidently the foci have been reached.

The efficacy of the intraspinal injections has been again attested to, lately, by Nebendahl,[16] who, among twelve cases so treated, had a mortality of 30 per cent., which compares very favorably with the high mortality of 80 per cent. among a small group treated without antitoxin.

Mitchell and Reilly [17] report meningococcus meningitis in a 4-months-old infant, cured by cistern puncture. Heist and Cohen [18] present evidence that the spinal fluid strains of meningococci are much more virulent for men than are the carrier strains.

The often striking difference between the severity of the clinical picture and the degree of infection or the number of meningococci in the lumbar puncture fluid is explained by Shearer and Parson [19] by the degree of acidosis of spinal fluid. A subnormal quantity of substances from which the cocci form acid or the presence of a strain of organisms less capable of producing the usual amount of acid may account for

13. Lewkowicz, K.: Arch. de méd. d. enf. **24**:329, 407, 549, 1921.
14. Woringer, P.: Arch. de méd. d. enf. **24**:159 (March) 1921.
15. Howell, W. W., and Cohen, S. A.: Intraventricular and Subdural Serum Treatment of Meningococcus Meningitis in Infancy, Am. J. Dis. Child. **24**:427 (Nov.) 1922.
16. Nebendahl: Arch. f. Kinderh. **71**:294 (Aug.) 1922.
17. Mitchell, A. G., and Reilly, J. J.: Am. J. M. Sc. **164**:66 (July) 1922.
18. Heist, G. D.; Cohen, S. S., and Cohen, M. S: J. Immunol **7**:1 (Jan.) 1922.
19. Shearer, C., and Parson, T. R.: Quart. J. Med. **14**:120 (Jan.) 1921.

the slight change in reaction of the patient's spinal fluid, even in the presence of a great number of meningococci; and, thus, for the mild clinical course.

Various Other Forms of Meningitis.—Strains of parameningococcus were isolated by Monserrat [20] in two sporadic cases and Cicszynski [21] describes purulent meningitis caused by typhoid bacillus, with entirely clear lumbar puncture fluid. Neumann and Kempner [22] and Fonzo [23] saw fatal meningitis due to streptotrix. The same micro-organism gave rise to a granulomatous tumor of the pons in a girl, reported by Zibordi.[24]

The presence of Kernig's sign in a surprisingly large proportion of infants and children with ordinary acute infections of nose and throat is interpreted by Göppert [25] as evidence of some involvement of the meninges, which may explain the restlessness or apathy of the patient. Loewy,[26] after studying 350 cases of serous meningitis with 425 lumbar punctures, confirmed the fact that the spinal fluid under normal conditions is a secretion, but in disease has more the character of a filtrate. The term serous meningitis expresses too much in some cases and not enough in others. In all infectious diseases with disturbances of the nervous system, acute serous meningitis is present, ranging from a mere toxic irritation of the vessels to infiltration of the pia, recognizable by lumbar puncture with proper interpretation.

Meningitis as a manifestation of mumps infection, although almost unknown in Buenos Aires, is rather common in Montevideo, not far away, according to reports of Morquis.[27] He observed a number of cases in which the meningitis preceded, accompanied or followed the parotitis; and in some instances meningitis was the only manifestation of the infection. Complete recovery was the rule, though sometimes deafness followed.

Tuberculous Meningitis.—The respiratory anomalies in tuberculous meningitis were studied by Eckstein and Rominger.[28] One group is due to the general miliary dissemination with its pulmonary and hematogenic factor. The other has its origin in the cerebrospinal

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20. Monserrat, C.: J. Philippine Islands M. A. **2**:15 (Jan.-Feb.) 1922.
21. Cicszynski, F. K.: Pedjatrja Pol. **2**, 1922.
22. Neumann, R., and Kempner, L. R.: Ztschr. f. klin. med. **94**:215 (June) 1922.
23. Fonzo, F.: Pediatria **30**:1045, 1922.
24. Zibordi, F.: Clin. Pediat. **3**:1, 1921.
25. Göppert: Klin. Wchnschr. **1**:64 (Jan. 8) 1922; abstr. **78**:930 (March 25) 1922.
26. Loewy, R.: Wien. Arch. f. inn. Med. **4**:305, 1922; abstr. J. A. M. A. **79**:1888 (Nov. 25) 1922.
27. Morquis, L.: An. de Fac. de med., Montevides **7**:23 (March-April) 1922.
28. Eckstein, A., and Rominger, E.: Arch. f. Kinderh. **70**:258 (Jan.) 1922.

affection itself and results from (1) irritation; (2) reflectoric inhibition, or (3) partial or total paralysis of the respiratory center. The manifestations consist in alterations of frequency and rhythm. They occur with a certain regularity in all three stages, especially at the end of the second and during the entire third stage. The first sign is the appearance of yawning and sighing; then follows acceleration, the irritative form, which changes into the periodic form, until this becomes of the Cheyne-Stokes type. These disturbances present a dissociation of the respiration on a functional basis.

A rare case of tuberculous meningitis with marked thickening of the spinal meninges from a diffuse infiltration with tubercles, extending from the cervical to the lumbar region, is reported by Regan and Cheney.[29] The findings explained the failure to obtain any spinal fluid by lumbar puncture.

The value of Rinda's shoulder sign for the early recognition of tuberculous meningitis has been confirmed by Tronconi.[30] The sign consists of a sudden movement of the shoulder when the head is briskly turned passively to the other side. Harbitz,[31] writing on the curability of tuberculous meningitis, points to the interesting fact that, while about 60 per cent. of all cases occur in the first two years of life and quite regularly result in death, the cases of recovery have occurred in older children and adults. Cramer and Bickel,[32] who report a recovery from this disease in a young man of 19, collected forty-four cases of recovery, in which 30 per cent. of the patients were over 20 and 2.5 per cent. under 2½ years of age.

In two cases a specific reaction, resulting from intradermal injection of the patient's own cerebrospinal fluid, was obtained by Korbsch.[33]

Artificial Pneumorachis.—Zorrillo y Polanco [34] thinks that subcutaneous injection of cerebrospinal fluid drawn by lumbar puncture, supplemented with injections of warm air into the spinal canal, may improve the prognosis of tuberculous meningitis. Injection of air or oxygen into the subarachnoid space by means of lumbar puncture was used by Sharp [35] in a few cases of epidemic meningitis, to render the evacuation of cerebrospinal fluid possible by mechanically breaking up secluded

29. Regan, J. C., and Cheney, G. W. H.: Tuberculous Meningitis with Dry Spinal Subarachnoid Due to Diffuse Tubercle Infiltration of Spinal Meninges, Am. J. Dis. Child. **22**:516 (Nov.) 1921.

30. Tronconi, S.: Pediatria, Naples **29**:802 (Sept.) 1921.

31. Harbitz, F.: Am. J. M. Sc. **161**:212 (Feb.) 1921.

32. Cramer, A., and Bickel, G.: Ann. de méd. **12**:226 (Sept.) 1922.

33. Korbsch, R.: Med. Klin. **17**:816 (July 3) 1921.

34. Zorrillo y Polanco, A.: Arch. españ. de pediat. **6**:462 (Aug.) 1922; abstr. J. A. M. A. **79**:2198 (Dec. 23) 1922.

35. Sharp, E. A.: Artificial Pneumorachis in Treatment of Acute Infections of Meninges, Arch. Neurol. & Psychiat. **6**:669 (Dec.) 1921.

pockets, or to drive out superfluous fluid before injecting serum. The results in sixty-four cases of various forms of meningitis justify a hopeful outlook for this class of meningococcus infection, in the opinion of the author.

HYDROCEPHALUS

As the cerebrospinal fluid, similarly to the blood or lymph, circulates in a closed vascular system, flowing from the ventricles through the foramina of Magendie and Luschka as the only paths of communication into the subarachnoid spaces of the cerebral sulci, where almost all of it is absorbed, an obstruction of the ventricles or of the larger subarachnoid spaces, the cisternae, must result in hydrocephalus; in the first case of the obstructive, in the latter of the communicative type. Dandy [36] holds it possible that hydrocephalus manifested soon after birth may be due to failure of the foramina to develop, or to intrauterine or postnatal inflammation. Adhesions following meningitis by occluding the cisternae are the most common cause of the communicative type. In every instance, the diagnosis of hydrocephalus and the exact site and character of obstruction can be determined by ventriculography and the phenolsulphonephthalein test, and the lesion can always be found at operation. The only satisfactory treatment of hydrocephalus is by attacking its cause directly; for instance by construction of a new foramen of Magendie. A similar attitude is maintained by Litchfield and Dembo,[37] who feel that the rôle of the endocrines in the hypersecretive form, while as yet indefinite, looms as a significant factor. The value of ventriculography for the differentiation of the type of hydrocephalus was demonstrated by E. B. Towne [38] in three cases. One of them presented the rare condition of unilateral obstructive hydrocephalus. The tendency of hydrocephalus in chondrodystrophia to spontaneous cessation of increase has been stressed by Dandy,[39] who attempts to explain this form by certain bone anomalies in the base of the skull, which during the process of growth disappear, the cause of hydrocephalus thus being eliminated.

Intravenous injections of a strongly hypertonic saline solution caused a brief initial rise of the intraventricular pressure, followed by marked depression in cats, in which Nañagas [40] had produced artificial hydrocephalus by injecting a suspension of lampblack into the lateral

36. Dandy, W. E.: Surg. Gynec. & Obst. **32**:112 (Feb.) 1921; Bull. Johns Hopkins Hosp. **32**:67 (March) 1921.

37. Litchfield, H. R., and Dembo, L. H.: Clinical and Therapeutic Aspects of Chronic Internal Hydrocephalus, J. A. M. A. **78**:711 (March 11) 1922.

38. Towne, E. B.: Value of Ventriculograms in Localization of Intracranial Lesions, Arch. Surg. **5**:144 (July) 1922.

39. Dandy, W. E.: Bull. Johns Hopkins Hosp. **32**:5 (Jan.) 1921.

40. Nañagas, J. C.: Bull. Johns Hopkins Hosp. **32**:381 (Dec.) 1921.

ventricles or into the cisterna cerebellomedullaris. This depression was possibly due to a rapid resorption of cerebrospinal fluid. Hypotonic fluids (distilled water) had the opposite effect. He also demonstrated that the absorption takes place through the ependymal into the underlying capillary network. The choroid plexus does not participate in this process. Wislocki and Putnam,[41] in their animal experiments, came to identical conclusions relative to absorption.

The production and absorption of the cerebrospinal fluid, etiologic pathology, symptomatology, ventriculography, principles of treatment and technic of operation are discussed in detail by Fraser and Dott.[42] In five cases of the extraventricular type, they have, by ligature of the common carotids, achieved promising results. Among thirteen cases of the ventricular variety, apparent arrest of the affection resulted from surgery in three instances, and a complete cure in two cases.

Cassel [43] found the prognosis of syphilitic hydrocephalus as a rule unfavorable despite energetic antisyphilitic treatment, as most patients retain more or less mental defects, and others become markedly neuropathic. Nevertheless, he urges energetic specific therapy in every case.

The pseudohydrocephalic complex in prematurely born infants, termed by Ylppoe megalocephalus, has been discussed by Rosenstern [44] in a paper on the secondary stigmata of prematurely born infants. He argues that the disproportion between the large head and the small body is due to an increase of the brain volume faster than that of the capacity of the skull. The intraspinal pressure was in every instance increased, but in no case did he find hydrocephalus in the postmortem examination. Megalocephalus is a partial manifestation of the peculiar somatic and psychic habitus of prematurely born infants, and is probably due to imperfection of their endocrine system and the premature loss of maternal hormones.

DEFECTIVE DEVELOPMENT

An elaborate work on the morphology of microcephaly has been contributed by Patane.[45] He gives exact craniometric data and a special description of the lower jaw of a 19-year-old microcephalic idiot with spastic tetraplegia, typical contractures, epilepsy and a marked development of lanugo. The principal features of the brain were porencephaly, microcephaly, simple hypotrophy, hydrocephalus (*ex vacuo*), atrophy of the corpus callosum and hypoplasia of the pyramidal tracts. The atrophic parts seem to be the site of the primary disturbance, the

41. Wislocki, G. B., and Putnam, T. J.: Am. J. Anat. **29**:313 (Sept.) 1921.
42. Fraser, J., and Dott, N. M.: Brit. J. Surg. **10**:165 (Oct.) 1922.
43. Cassel: Deutschr. med. Wchnschr. **48**:655 (May 19) 1922.
44. Rosenstern, J.: Ztschr. f. Kinderh. **32**:129 (April) 1922.
45. Patane, M.: Riv. di antrop. **24**:209, 1921.

hypotrophic ones being probably associated areas, depending on the former. The microgyria is considered as due to inflammatory processes. Interesting conclusions are made as to the development of the fissura Sylvii, and, in the assumption of certain laws of development, as to a mutual influence of adjacent parts.

Partial absence of the corpus callosum in a 15-year-old idiot is described by Cameron and Nicholls,[46] with a detailed clinical picture and complete necropsy report; and congenital hydranencephaly due to inflammatory processes and abnormalties of the vascular "anlage" with an almost complete absence of brain and midbrain in an intact skull by Licen.[47]

Young [48] reports in full two acrocephalic cases, and after a review of the various conjectures relative to the etiology, argues in favor of dysendocrinism dating from the blastoma stage being the basis of the condition. Weygandt,[49] in a minority of cases of oxycephaly, found weakmindedness or degenerative features and psychotic symptoms.

Blechmann [50] cites two cases of homalopsia exophthalmica, a congenital dystrophy of the skull, possibly of syphilitic origin, and characterized by a markedly flattened face, retraction of the bridge of nose, deformity of the palate and exophthalmus. The anomaly is associated with retarded dentition and psychic defects, and has a certain resemblance to mongolian idiocy and scaphocephalus. Apert classifies the condition among the syndromes of craniosynostosis, an anomaly of cranial ossification.

A spheno-orbital meningocele in a 2-year-old boy, of the size of the child's head, has been reported by Lutz.[51] Death from meningitis followed operation. The author compares with this the cases of facial encephalocele on record. The mortality ranged from 20 to 31 per cent. Intranasal transsphenoid encephalocele in a boy of 6 is described by Nager.[52] Death resulted one year later from streptococcus meningitis. The author was able to find only six analogous instances on record. A dicephalus is described by Beach.[53]

A contribution to the etiologic problem of porencephalus is offered by Globus.[54] He gives a detailed histopathologic description, with

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46. Cameron, J., and Nicholls, A. G.: Canadian M. A. J. **11**:448 (June) 1921.
47. Licen, E.: Ztschr. f. ges. Neurol. u. Psych. **64**:282, 1921.
48. Young, F. W.: Arch. Pediat. **39**:629 (Oct.) 1922.
49. Weygandt: Deutsch. Ztschr. g. Nervenh. **68-69**:495, 1921.
50. Blechmann, G.: Bull. Soc. de pédiat. de Paris **9**:403, 1922.
51. Lutz, F.: Brazil-med. **35**:17 (Jan. 8) 1921; abstr. J. A. M. A. **76**:899 (March 26) 1921.
52. Nager, F. R.: Schweiz. med. Wchnschr. **52**:516 (May 25) 1922.
53. Beach, W. M.: Case of Diredydimus (Dicephalus), J. A. M. A. **76**:1748 (June 18) 1921.
54. Globus, J. H. B.: Contribution to the Histopathology of Porencephalus, Arch. Neurol. & Psychiat. **6**:652 (Dec.) 1921.

numerous illustrations of the porencephalic brain of an 11-months-old child. The defect proved to be due to meningo-encephalitis, leading to strangulation of the blood vessels with subsequent sclerotic changes. The findings are important in relation to the pathology of disseminated sclerosis.

Spina Bifida.—The roentgenographic examination of the vertebra and its congenital anomalies in the lumbosacral region is important and has revealed a great number of conditions hardly suspected. Hintze,[55] in a classic paper, deals with the relation of the normal sacral fontanel (fontanella lumbosacralis) to the pathologic spina bifida occulta and their differentiation in the roentgenogram. He details the findings in 150 skeletons of anthropoid apes; 400 human sacrums and lumbar vertebrae, and the roentgenograms of 700 others. He discusses the observations on the symptom complex of lumbosacral hypertrichosis, neuroparalytic disturbances in the lower extremities and vertebral hiatus (morbus diaboli) and myelodysplasia (Fuchs).

The anatomicopathologic conditions in spina bifida occulta which bring on progressive deformities of the feet, with a tendency in the majority of cases toward the development of pes equinus, varus, excavatus and their variations and combinations, as well as the principles of treatment, are dealt with by Roeren.[56] There are certain features in the minute structure in spina bifida that have a direct bearing on operative procedures, on prognosis as regards nervous function after and apart from operation; these have been detailed by Keiller.[57]

The remarkable occurrence of some degree of spinal defect in four children has been recorded by Pybus,[58] namely, spina bifida, spina bifida occulta, meningomyelocele in the sacral region and an exaggerated postanal dimple.

Valuable data relative to the frequency of heredofamilial defects, various localizations, types, cord involvement, clinical signs and operative results are presented by Woltman,[59] who reviews 187 cases of spina bifida, including three cases of myelodysplasia without bone defects. The opinions relative to the rôle spinal malformations, especially spina bifida occulta, play as factors in enuresis are still contradictory. That an anatomic basis for enuresis is not so rare as supposed is claimed by Sironi and Sabatini.[60] In their series of twenty cases of enuresis in children, in sixteen the fifth vertebra was deformed or there was spina

55. Hintze, A.: Arch. f. klin. Chir. **119**:409 (April) 1922.
56. Roeren, L.: Arch. f. orthop. Unfallschir. **19**:1-49, 1921.
57. Keiller, Violet H.: Brain **45**:31 (June) 1922
58. Pybus, F. C.: Lancet **2**:599 (Sept. 17) 1921.
59. Woltman, H. W.: Minnesota Med. **4**:244 (April) 1921.
60. Sironi, L., and Sabatini, L.: Riv. di clin. pediat. **20**:399 (July) 1922; abstr., J. A. M. A. **79**:1644 (Nov. 4) 1922.

bifida; and in one case the fifth vertebra and the sacrum had separated. In some, there was also myelodysplasia. No deformities of this kind were encountered in other children, free from enuresis. Peritz, quoted by Woltman,[59] has found 68 per cent. of spina bifida occulta among twenty-two adults with enuresis and 35 per cent. of spina bifida among twenty-two children with enuresis. Systematic studies by Altschul [61] of vertebrae of patients with enuresis revealed, in about three-fourths of the cases, anomalies of vertebrae. A number of rare malformations were discovered, as various types of lateral fissures, defects of the corpus vertebrae, spina bifida anterior, supernumerous rudiments and wedge-shaped vertebrae with supernumerary ribs. Chute [62] thinks that spina bifida occulta may explain a number of perplexing bladder disturbances. Operations have, on the whole, not been very satisfactory. The outlook, he states, would probably be more hopeful, if the operation were to be performed earlier.

On the other hand, Bonsmann,[63] who found spina bifida not only in families with enuresis but also in healthy families, failed to establish a connection between anomalies of vertebra and enuresis; and Gelpke [64] opposes Fuch's attempt to bring enuresis in an etiologic relationship to myelodysplasia.

Two cases of agenesis (congenital paralysis) of cranial nerves are reported by Cadwalader.[65]

Morgan and Stuart [66] report Oppenheim's disease in a boy of 9, in whom the weakness was limited to the lower extremities, with a tendency to marked improvement. They state that approximately 125 instances have been reported in the twenty years since Oppenheim described the affection. Some authors assume a close relationship between Oppenheim's disease and Werdnig-Hoffmann's affection. Leenhardt and Sentis,[67] for instance, who review the literature on these anomalies and analyze two of their own clinical observations, conclude that these diseases may be of the same nature and should be included under the nosologic term of diffuse chronic poliomyelitis of early infancy. In a contribution to the problem of the so-called congenital encephalitis of Virchow, stressing the subject of cortical and medullary encephalomalacia and sclerosis, Wohlwill [68] describes minutely nine cases, and

61. Altschul, W.: Fortsh. a. d. geb. d. Roentgenstrahl. **27**:607, 1921.
62. Chute, A. L.: J. Urol. **5**:317 (April) 1921.
63. Bonsmann, M. R.: Deutsch. Ztschr. f. Nervenh. **74**:343, 1921.
64. Gelpke, H.: Monatschr. f. Kinderh. **19**:137 (Nov.) 1921.
65. Cadwalader, W. B.: Am. J. M. Sc. **163**:744 (May) 1922.
66. Morgan, W. G., and Stuart, D. D. V., Jr.: Amytonia Congenital Complicated by Acute Cholecystitis of Typhoid Origin, J. A. M. A. **78**:1105 (April 15) 1922.
67. Leenhardt and Sentis: Arch. de méd. d. enf. **24**:137 (March) 1921.
68. Wohlwill: Ztschr. f. ges. Neurol. u. Psychiat. **73**:360, 1921.

comes to the conclusion, arrived at also by Schwartz,[69] that Virchow's interstital congenital encephalitis cannot be considered encephalitis in the strict sense of the term.

Comby[70] relates his experiences with sixty-two cases in children suffering from acute encephalitis in connecetion with influenza, whooping cough, enteritis, measles and syphilis or from forms apparently having no connection with any other disease; and reviews the wide range of the symptoms. Recovery without sequelae ensued in one third of his cases.

Griffith[71] analyzes thirty-one cases of acute cerebellar encephalitis with two necropsies. In one fourth of the cases, there were signs of mental defects.

Hemorrhagic encephalitis following appendicitis with fibropurulent peritonitis recurred about ten years later in connection with influenzal pneumonia in a boy whose case is reported by Heusser.[72] These two successive brain processes were found in postmortem examination.

LETHARGIC OR EPIDEMIC ENCEPHALITIS

No single topic in the field of neurology has in the past few years received more attention than epidemic encephalitis and its sequelae. Only papers dealing with the disease in childhood can be covered in this brief review, though others will be given a passing consideration.

In the 274 cases studied by Josephine B. Neal[73] half of the patients were 15 years of age or younger; and 50 per cent. more boys were affected than girls, as is the case in epidemic meningitis and poliomyelitis. The symptoms in children did not differ greatly from those in the adults; the onset was more frequently sudden and the course ordinarily shorter than in the adults. In addition to the symptoms of infection, such as fever, headache, vomiting, constipation and malaise, those directly referable to the central nervous system showed an almost endless variety, comprising lethargy or insomnia, convulsions, ocular disturbances, paralyses of wide distribution, especially of the cranial nerves, disturbances of speech, twitchings, tremor, choreiform movements, catatonia, various changes in reflexes, and also in the Babinski's sign, and ankle clonus and profuse sweating. Typical meningeal signs were not common. The mortality in her series was 28 per cent.

Two well defined periods of the affection are distinguished: the acute stage and the sequelae. The polymorphous clinical pictures of

69. Schwartz: Ztschr. f. Kinderh. **29**:102; **31**:51, 1921.
70. Comby, J.: Arch. de méd. d. enf. **24**:457, 488 (Aug.) 1921.
71. Griffith, J. P. C.: Am. J. M. Sc. **162**:781 (Dec.) 1921.
72. Heusser, H.: Deutsch. Ztschr. f. Nervenh. **71**:210, 1921.
73. Neal, Josephine B.: Experience with More than 100 Cases of Epidemic Encephalitis in Children, J. A. M. A. **77**:121 (July 9) 1921.

the first stage are classified by Hofstadt [74] in the following groups: (1) the pure encephalitic types with a choreic, myoclonic, lethargic and choreic-athetotic subform; (2) the type resembling Landry's paralysis, with fatal issue; (3) the meningeal form, with typical meningeal symptoms; (4) the myelitic form; (5) the frequent abortive forms, under the mask of a slight "grippal" infection. Such abortive cases are especially discussed by Wallgren,[75] and two ambulatory cases are discussed by Williams.[76]

With the wide range of and the manifold combinations in the symptomatology, the differential diagnosis, as discussed by Barker [77] minutely, may include a number of infections and intoxications, which simulate this disease: syndromes due to vascular lesions, intracranial neoplasms, chorea. parkinsonian disease, cataleptic and catatonic states, myasthenia gravis, progressive muscular atrophy, hysteria and neurasthenia. Yet despite the numerous clinical forms, the somnolent-ophthalmoplegic, the paralytic, amyostatic and hyperkinetic pictures are the commonest.

Two other important inflammatory, nonsuppurative diseases of the central nervous system which have certain features in common, namely syphilis and poliomyelitis, may give rise to occasional difficulties in differentiation. Bassoe [78] points to the apparent existence in encephalitis (as in syphilis) of a para stage, a progressive cerebral process coming on sometimes long after the acute illness, the virus giving rise to chronic and degenerative processes.

An interval as long as one and one-half years between the acute stage and recurrence is reported by Price; [79] and recrudescence after one year is reported by Blakeslee.[80]

An extremely malignant, rapidly spreading ward epidemic has been observed by Duzar and Baló [81] in which eleven babies contracted the disease. Nine died, of whom seven were examined postmortem. There were symptoms similar to those in alimentary intoxication, fever, convulsions, jactitation, disturbances of the sensorium, toxic respiration, hypertonia and ocular symptoms, the diagnosis proving very difficult clinically. The resemblance to severe alimentary intoxication in infants

74. Hofstadt, F.: Ztschr. f. Kinderh. **29**:190 (June) 1921.
75. Wallgren, A.: Deutschr. med. Wchnschr. **47**:1053 (Sept. 8) 1921.
76. Williams, C. E.: Lancet **2**:499 (Sept. 3) 1921.
77. Barker, L. F.: Diagnostic Criteria in Encephalitis and Encephalomyelitis, Arch. Neurol. & Psychiat. **6**:173 (Aug.) 1921.
78. Bassoe, Peter: Diagnosis of Encephalitis, J. A. M. A. **79**:2223 (Dec. 30) 1922.
79. Price, G. E.: Encephalitis; Recurrence of Symptoms One and One-Half Years After Apparent Recovery, J. A. M. A. **78**:716 (March 11) 1922.
80. Blakeslee, G. A.: Neurol. Bull. **3**:159, 1921.
81. Duzar, J., and Baló, J.: Jahrb. f. Kinderh. **99**:209 (Sept.) 1922.

is also emphasized by Voigt [82] and others, and the prevalence of local and general convulsions causing difficulty in diagnosis is mentioned by Stadelmann.[83] A diagnostic aid was the fact that Pandy's reaction was almost always negative, though lymphocytosis existed frequently.

In twenty-eight cases analyzed by Moore [84] 80 per cent. of the patients were somnolent and 20 per cent. died. The rare occurrence of the disease in a new-born has been recorded by Hoobler.[85]

Kernig's and Brudzinski's signs, as noted by various writers, for example Bardach,[86] were missing even in meningeal irritation, this serving as a valuable differential diagnostic factor as regards tuberculous meningitis. The sketches given by Bardach of the symptomatology, as noted by her during the epidemic in Dusseldorf (1920 and in 1921), make the complexity of the clinical picture again apparent. Cases were seen with the picture of dementia praecox, glycosuria, striking relapses and marked intestinal contractions. Massari had described similar contractions of the ileum and cecum of such violent character that a surgical intra-abdominal affection was suspected and laparotomy performed; and in the three cases of epidemic encephalomyelitis reported by Bolaffio,[87] intense abdominal pains suggested appendicitis or ileus. The author adds that in similar cases operation has been performed, with negative findings after laparotomy.

Of the twenty-seven cases reported by Palitzsch [88] the majority represented the hypertonic akinetic type, frequently in combination with tremor, ocular paralysis and lethargy; the minority representing the atonic hyperkinetic form with muscular atonia, choreic or athetotic movements and myoclonia.

Purely spinal types are discussed by Riley.[89] Symptomatically, they are divided into two groups: (1) ventral poliomyelitis, with the irritative and paralytic subgroups; and (2) the transverse myelitic type. There are transitional forms.

Sequelae.—The greater our experience with the disease, the more are we impressed by the frequency and seriousness of its sequelae, which are so characteristic that they allow a retrospective diagnosis of the nature of the previous acute disease.

With the exception of one, the forty-five patients observed by Hofstadt [90] who were discharged from hospital as cured after the

82. Voigt, O.: Monatschr. f. Kinderh. **21**:156 (May) 1921.
83. Stadelmann, E.: Monatschr. f. Kinderh. **19**:294, 1921.
84. Moore, C. U.: Northwest Med. **20**:176 (July) 1921.
85. Hoobler, Raymond: Arch. Pediat. **38**:461, 1921.
86. Bardach, M.: Monatschr. f. Kinderh. **22**:475 (Dec.) 1922.
87. Bolaffio, A.: Riv. crit. di clin. med., July 15, 1922, p. 232.
88. Palitzsch, F.: Deutsch. Arch. f. klin. Med. **135**:115 (Jan.) 1921.
89. Riley, H. A.: Spinal Forms of Encephalitis, Arch. Neurol. & Psychiat. **5**:408 (April) 1921.
90. Hofstadt, F.: Ztschr. f. Kinderh. **29**:272 (June) 1921.

acute stage proved later not to be cured. After a free interval of from one to nine weeks, they manifested various morbid symptoms of a striking character, then new to the pediatrician. Among the polymorphous syndromes of the sequelae, five principal characteristic groups could be discerned: 1. Agrypnia, the most frequent and extremely intractable, extending over months, characterized by a postponement of falling asleep and often also by reduction of the total sleeping time per day, and accompanied frequently by excessive nocturnal hyperkinesis of a great variety of types. 2. The amyostatic syndromes. 3. Chronic chorea and chorea-athetosis, a syndrome considered a manifestation of the extrapyramidal system. 4. The psychic disturbances of various forms, graded from slight alterations of personality or psychic transformations to marked hypomanic conditions; from mild to severe degrees of dementia and ethical defects. Flight of ideas, marked lack of control, exaggerated euphoria, psychomotor hyperactivity, destructive tendencies, lack of power to cencentrate, quarrelsomeness, talkativeness were encountered. The conditions often reminded one of the psychoses of manic character (hypomania). In another group of children, the psychic transformations were of a depressive type or the features had a semblance of hysteria or neuropathia. 5. Adiposogenital complex.

Agrypnia seems to be capable of improvement eventually, while the amyostatic complexes probably are irreparable and in the course of years lead to dementia and death.

About one fourth of the cases of Price [91] terminated fatally, and the remainder were left with persistent sequelae; and Grossman,[92] among ninety-two patients, adults and children, discovered in sixty-two a more or less progressive involvement of the central nervous system, with forty-two of them manifesting a parkinsonian syndrome and various psychic disturbances. In the experience of Barker [93] complete recovery was the exception, perhaps 75 per cent. of the cases exhibiting residual phenomena as hyperkinetic, hyperesthetic and paresthetic syndromes, autonomic disorders, disturbances of coordination, pupillary and ciliary disturbances, sialorrhea and a number of minor and major psychoses. A considerable number of children were rendered permanently deficient mentally.

The great frequency of parkinsonian residues observable in France during the last two years has been commented upon by Laignel-Lavastine.[94]

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91. Price, G. E.: Am. J. M. Sc. **163**:871 (June) 1922.

92. Grossman, M.: Sequels of Encephalitis, J. A. M. A. **78**:959 (April 1) 1922.

93. Barker, L. F.: Sequels of Epidemic (Lethargic) Encephalitis and Their Treatment, J. A. M. A. **78**:1417 (May 6) 1922.

94. Laignel-Lavastine: Médicine **3**:325 (Feb.) 1922.

It is especially the nocturnal wakefulness and diurnal somnolence, the inversion of the rhythm of sleep that forms the subject of numerous papers. Rolleston and Fletcher,[95] Progulski and Groebel,[96] Rutimeyer,[97] Reh,[98] Hofstadt,[99] Janecke,[100] Franconi,[101] Roasenda,[102] Zalla [103] and others have reported such serious sleep disturbances, often associated with delirious choreic states, restless agitation with singing, whistling, tossing from side to side, shouting and talking; partly also in association with changes of character and mind.

Parker [104] analyzes striking disorders of the respiratory rhythm, or constant dyspnea, Aronson [105] describes tachyrespiria, and Haas [106] describes attacks of forced breathing (hyperpnea) that invariably ended in the pseudotetany syndrome.

Precocious sexual feelings and intense eroticism were observed by Leahy and Sands.[107] Kirschbaum [108] reports asocial tendencies, brutality and moral insanity. Bonhoeffer [109] has similar experiences. Kauders [110] studying the large material of the Wagner Jauregg clinic at Vienna, saw repeatedly moria-like mental states with silly cheerfulness, desultory thinking, tendency to punning, euphoric dementia, intellectual defects and cases of extreme psychomotor restlessness, "hyperkinetic motility psychoses," and stereotyped movements. Similar experiences were met by Staehelin [111] in Zürich, Hohman,[112] Paterson and Spence,[113] and Cruchet.[114] Burt [115] gives a synopsis of the English publications on psychic sequelae, with the purpose of bringing this etiologic factor in antisocial behavior to the attention of those who deal with the criminality of children.

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95. Fletcher, H. M.: Brit. J. Dis. Child. **18**:69 (April-June) 1921.
96. Progulski, S., and Groebel, A.: München. med. Wchnschr. **68**:451 (April 15) 1921.
97. Rutimeyer, W.: Schweiz. med. Wchnschr. **51**:7 (Jan. 6) 1921.
98. Reh, T.: Rev. med. de la Suisse Rom. **41**:184 (March) 1921.
99. Hofstadt, F.: München. med. Wchnschr. **67**:1400 (Dec. 3) 1920.
100. Janecke, A.: Deutsch. med. Wchnschr. **46**:1388 (Dec. 9) 1920.
101. Franconi, C.: Policlinico **28**:575, 1921.
102. Roasenda: Policlinico **28**:181 (Feb. 17) 1921.
103. Zalla: Riv. di patol. nerv. **25**:375, 1920.
104. Parker, H. L.: Disturbances of Respiratory Rhythm in Children, Arch. Neurol. & Psychiat. **8**:630 (Dec.) 1922.
105. Aronson, L. S.: Neurol. Bull. **3**:113, 1921.
106. Haas, S. V.: Arch. Pediat. **38**:785, 1921.
107. Leahy, S. R., and Sands, I. J.: Mental Disorders in Children Following Encephalitis, J. A. M. A. **76**:373 (Feb. 5) 1921.
108. Kirschbaum, M.: Ztschr. f. ges. Neurol. u. Psychiat. **73**:599, 1921.
109. Bonhoeffer, K.: Klin. Wchnschr. **1**:1446 (July 15) 1922.
110. Kauders: Ztschr. f. ges. Neurol. u. Psychiat. **74**:431, 1922.
111. Staehelin, J. E.: Ztschr. f. ges. Neurol. u. Psychiat. **77**:171, 1922.
112. Hohman, L. B.: Bull. Johns Hopkins Hosp. **33**:372 (Oct.) 1922.
113. Paterson, D., and Spence, J. C.: Lancet **2**:491 (Sept. 3) 1921.
114. Cruchet, R.: J. de méd. de Bordeaux **92**:20 (Jan.) 1921.
115. Burt, C.: Brit. J. Psychol. **2**:237 (April) 1922.

Duverger and Barré [116] state that various ocular manifestations must be anticipated for years to come and should not be mistaken for neurosyphilis.

Among the rare consequences of epidemic encephalitis, hypophysial syndromes with adiposity and genital atrophy are reported by Stiefler,[117] Kauders,[110] Mayer,[118] Bychowski,[119] Bertolani,[120] Barkman [121] and others; obesity, by Livet; [122] myasthenia gravis and progressive lipodystrophy, by Sarbó; [123] pubertas praecox by Stern; [124] a very oily seborrhea of the face (Salbengesicht) by Stiefler [125] in an adult.

Spinal Fluid.—The conditions of the spinal fluid have been described at length by Kraus and Pardee [126] and Eskuchen.[127] The latter, summarizing the available data, states that two spinal fluid syndromes are to a certain degree characteristic: (1) pleocytosis, globulin increase, "luetic" gold curve and hyperglycorrhachia; (2) the triad: cytoglobulin dissociation (high cell count in proportion to the amount of globulin) "luetic" gold curve and hyperglycorrhachia. Other contributions were made by Thalhimer [128] and Foster [129] on hyperglycorrhachia; by Howell,[130] Davis and Kraus,[131] Happ and Mason,[132] on the colloidal gold reaction. The presence of an antibody was demonstrated by Quest [133] by means of intradermal autoseroreaction.

Pathologic Anatomy.—The histopathologic studies by Siegmund [134] in twenty-one cases revealed the typical pictures, characterized by

116. Duverger and Barré: Bull. méd., Paris, 1921, p. 361.
117. Stiefler: Monatschr. f. Psych. u. Neurol., August, 1921.
118. Mayer, W.: München. med. Wchnschr. **68**:552 (May 6) 1921.
119. Bychowski, Z.: Ztschr. f. ges. Neurol. u. Psychiat. **76**:508, 1922.
120. Bertolani: Riv. sper. de freniat. **45**:467, 1922.
121. Barkman, A.: Acta med. Scandinav. **56**:188 (March) 1922.
122. Livet, L.: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:656 (May 6) 1921.
123. Sarbó: Orvosi hetil. **65**, 1921; Deutsch. Ztschr. f. Neurol. **74**:285, 1922.
124. Stern, F.: Med. klin. **18**:864 (July 2) 1922.
125. Stiefler: Ztschr. f. Neurol. u. Psychiat. **73**:455, 1921.
126. Kraus, W. M., and Pardee, I. H.: Serology of Spinal Fluid and Blood in Encephalitis, Arch. Neurol. & Psychiat. **5**:710 (June) 1921.
127. Eskuchen, K.: Ztschr. f. ges. Neurol. u. Psychiat. **76**:568, 1922.
128. Thalhimer, William, and Updegraff, Helen: Sugar Content of Blood and Spinal Fluid in Epidemic Encephalitis, Arch. Neurol. & Psychiat. **8**:15 (July) 1922.
129. Foster, H. E.: Hyperglycorachia in Encephalitis, J. A. M. A. **76**:1300 (May 7) 1921.
130. Howell, K. M.: Colloidal Gold Curve in Encephalitis, Arch. Neurol. & Psychiat. **7**:229 (Feb.) 1922.
131. Davis, T. K., and Kraus, W. M.: Am. J. M. Sc. **161**:109 (Jan.) 1921.
132. Happ, W. M., and Mason, V. R.: Bull. Johns Hopkins Hosp. **32**:137 (May) 1921.
133. Quest, R.: Jahrb. f. Kinderh. **96**:324, 1921.
134. Siegmund, H.: Frankfurt. Ztschr. f. Pathol. **25**:526, 1921.

perivascular round and plasma cell infiltration, nodular or diffuse proliferation of glia cells, degenerative processes in the ganglion cells and neuronophagia. The site of predilection is the gray matter around the aquaeductus, the third ventricle and the lateral ventricles, without sparing entirely, however, other parts of the brain, including the white matter. In seven cases, there were meningitic changes; in three cases the spinal cord was involved; and in six cases, a streptococcus was isolated. Pathologically and anatomically, the affection is classified by the author in the group of poliomyelitis, typhus fever and trypanosomiasis (sleeping-sickness).

Minute pathologic findings are recorded by Grütter.[135] Da Fano [136] writes on the histopathology, and Lucksch,[137] on ganglion cells. The findings in a case of epidemic encephalitis with myelitis are detailed by Spiller.[138] Calcification of cerebral vessels is described by Dürck,[139] and the subject of disturbances of the liver in epidemic encephalitis is taken up by Stern and Meyer-Bisch.[140] Duzar and Balo [81] describe briefly the pathologic and histologic findings in seven infants.

Epidemiology, Etiology and Bacteriology.—An increasing number of publications speak in favor of the contagious nature of the disease. Kling,[141] who under the most favorable conditions studied the manner of propagation of the disease in a sparsely populated part of Lapland, could follow up direct transmissions from person to person. The incubation time was ten days, and the virus was demonstrated in the nasopharynx of well persons who had been in contact with the sick. Very frequent were the abortive cases with catarrhal symptoms predominating. Convincing evidences of contagiosity are farther recorded by Netter,[142] Stiefler,[143] Claude and de Laulerie,[144] Roger and Blanchard [145] and Pecori.[146] Placental transmission is reported by Mercier.[147] Investigations of the filtrable virus were carried on by

135. Grütter, E.: Ztschr. f. ges. Neurol. u. Psychiat. **72**:29, 1921.
136. Da Fano, C.: Brit. M. J. **1**:153 (Jan. 29) 1921.
137. Lucksch: Klin. Wchnschr. **1**:1528, 1922.
138. Spiller, W. G.: Encephalitis with Myelitis, Arch. Neurol. & Psychiat. **7**:739 (June) 1922.
139. Dürck, H.: Ztschr. f. ges. Neurol. u. Psychiat. **72**:179, 1921.
140. Stern, F., and Meyer-Bisch, R.: Klin. Wchnschr. **1**:1559 (July 29) 1922.
141. Kling: Norsk Mag. f. Lævidensk. **83**:843, 1922; Current Comment, J. A. M. A. **79**:1771 (Nov. 13) 1922.
142. Netter et al.: Bull. Acad. de méd. **85**:278 (March 8) 1921.
143. Stiefler: Ztschr. f. ges. Neurol. u. Psychiat. **74**:396, 1922.
144. Claude, H., and de Laulerie, J.: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:36 (Jan. 21) 1921.
145. Roger, H., and Blanchard, A.: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:40 (Jan. 21) 1921.
146. Pecori: Ann. d'ig. sper. **31**:32, 1921.
147. Mercier, R. et al.: Bull. Acad. de med. **85**:625 (May 31) 1921.

Netter, Césari and Durand,[148] Thalhimer,[149] Davide and Liljenquist,[150] Bastei,[151] Levaditi and Harvier[152] and others.

Economo[153] holds *Diplostreptococcus pleomorphus* of Wiesner (1917) as the causative agent, and he cites Gross and Reichhard of Heidelberg, who found this micro-organism in all their cases of epidemic encephalitis.

Rosenow[154] isolated a streptococcus of the green producing variety in a case of encephalitis, and also proved the infectious nature of epidemic[155] hiccup. His experimental results suggest a relationship between epidemic hiccup and encephalitis.

That epidemic encephalitis and poliomyelitis can be distinguished by the power of blood serum of patients convalescent from poliomyelitis to neutralize the virus of poliomyelitis, the serum of those convalescent from encephalitis being void of this power, has been demonstrated by the immunologic studies of Amoss.[156]

The finding by Hilgermann, Lauxen and C. Shaw[157] of parasites in the blood during a recurrence demonstrates that the disease may last a long time, the persistence of the parasite within the human body involving a permanent danger of propagation.

Treatment.—In the experience of Economo[153] the use of hexamethylenamin and injections of Pregl's iodin solution in large doses had striking results. Dattner[158] used successfully injections of Pregl's solution and an iodin preparation with typhoid vaccine. Helmholz and Rosenow[159] obtained striking results in three patients treated with serum from a horse repeatedly injected with streptococcus that had been isolated in a patient with typical encephalitis. Piticariu[160] used suc-

148. Netter, A.; Césari and Durand, H.: Bull. et mém. Soc. méd. d. hôp. de Paris **45**:751 (May 13) 1921.

149. Thalhimer, William: Encephalitis Cultural and Experimental Studies, Arch. Neurol & Psychiat. **5**:113 (Feb.) 1921.

150. Kling, C.; Davide, H., and Liljenquist, F.: Hygiea **83**:566 (Sept. 16) 1921; abstr., J. A. M. A. **77**:1613 (Nov. 13) 1921.

151. Bastei, P.: Arch. per le sc. med., Torino **44**:212, 1921.

152. Levaditi, C., and Harvier, P.: Compt. rend. Soc. de biol. **84**:300, 957, 1921; Ann. de l'Inst. Pasteur, 1922. Harvier, P.: Paris méd. **11**:485, 1921; abstr. J. A. M. A. **77**:495 (Aug. 6) 1921.

153. Economo: Wien. med. Wchnschr. **71**:1322, 1921.

154. Rosenow, E. C.: Etiology of Encephalitis, J. A. M. A. **79**:443 (Aug. 5) 1922.

155. Rosenow, E. C.: Diaphragmatic Spasms in Animals Produced with a Streptococcus from Epidemic Hiccup, J. A. M. A. **76**:1745 (June 18) 1921; Northwest Med. **21**:329 (Sept.) 1922.

156. Amoss, H. L.: J. Exper. Med. **33**:187 (Feb.) 1921.

157. Hilgermann, Lauxen, and Shaw, C.: Med. Klin. **18**:17 (Jan. 1) 1922.

158. Dattner, B.: Wien. klin. Wchnschr. **34**:351, 1921.

159. Helmholz, H. F., and Rosenow, E. C.: Encephalitis Treated with Specific Serum, J. A. M. A. **79**:2068 (Dec. 16) 1922.

160. Piticariu, I.: Wien. klin. Wchnschr. **35**:441 (May 11) 1922.

cessfully intravenous injections of the patient's own lumbar puncture fluid, which, he believes, may contain antibodies. Voigt [82] saw in two cases a striking subsidence of the symptoms regularly following repeated lumbar punctures, until recovery took place. Leahy and Sands [161] write on the management of children presenting postencephalitic syndromes, such as the reversal from nocturnal to diurnal sleep. Laignel-Lavastine,[94] Barré and Reys [162] and Radovici and Nicolesco [163] report some results from scopolamin and atropin in involuntary movements and in the parkinsonian syndrome. Cheinisse,[164] who reviews the different publications on treatment, asserts that none have stood the test of time except the turpentine fixation abscess, which reduces the mortality considerably.

The following contributions, though not exclusively dealing with the disease in childhood, are of interest for the pediatrician: the article of Hunt [165] on striatal and thalamic types; of Abrahamson [166] on motor disturbances; of Kirby and Davis; [167] of Jones and Raphael [168] on the psychiatric features of the disease; of Grossman [169] on late results; of Pearl [170] on statistical notes; of Alexander [171] on treatment; the Public Health Report [172] on world wide prevalence; of Kahlmeter,[173] Reeves,[174] Happ and Mason [175] on clinical studies; Mayer [176] on late conditions; Isserlin [177] on extrapyramidal disorders after encephalitis; of Mingazzini,[178] von Monakow [179] on morphology and pathogenesis, and a French committee report [180] on the history of the disease.

161. Leahy, S. R., and Sands, C. J.: New York M. J. **114**:166 (Aug. 3) 1921.
162. Barré, J. A., and Reys, L.: Paris méd. **11**:261 (Oct. 1) 1921; Bull. méd., Paris **35**:351 (April) 1921.
163. Radovici, A., and Nicolesco, J. F.: Presse méd. **29**:83 (Jan. 29) 1921.
164. Cheinisse, L.: Presse méd. **29**:146 (Feb. 19) 1921; abstr., J. A. M. A. **76**:967 (April 2) 1921.
165. Hunt, J. R.: Am. J. M. Sc. **162**:481 (Oct.) 1921.
166. Abrahamson, I.: Motor Disturbances in Encephalitis, Arch. Neurol. & Psychiat. **5**:33 (Jan.) 1921.
167. Kirby, G. H., and Davis, T. K.: Psychiatric Aspects of Epidemic Encephalitis, Arch. Neurol. & Psychiat. **5**:491 (May) 1921.
168. Jones, B. L., and Raphael, T.: Psychiatric Features of So-Called Encephalitis, Arch. Neurol. & Psychiat. **5**:150 (Feb.) 1921.
169. Grossman, M.: Late Results in Encephalitis, Arch. Neurol. & Psychiat. **5**:580 (May 1921.
170. Pearl, R.: Bull. Johns Hopkins Hosp. **32**:221 (July) 1921.
171. Alexander, A.: Deutsch. med. Wchnschr. **47**:1547, 1921.
172. Public Health Report **36**:602, 1921.
173. Kahlmeter, G.: Clinical Aspects of Encephalitis, München. med. Wchnschr. **68**:669 (June 3) 1921.
174. Reeves, R. S.: New York M. J. **114**:702 (Dec. 21) 1921.
175. Happ, W. M., and Mason, Y. R.: Bull. Johns Hopkins Hosp. **32**:137 (May) 1921.
176. Mayer, W.: München. med. Wchnschr. **69**:552 (May 6) 1921.
177. Isserlin, A.: Deutsch. Ztschr. f. Nervenh. **74**:319, 1922.
178. Mingazzini, G.: Zentralbl. f. ges. Neurol. u. Psychiat. **63**:199, 1921.
179. Von Monakow, C.: Schweiz. Arch. f. Neurol. u. Psychiat. **10**:3, 1922; abstr. Arch. Neurol. & Psychiat. **8**:299 (Sept.) 1922.
180. Bull. Acad. de méd. **85**:278, 1921.

CHOREA MINOR

The analysis of the chorea material at the Children's Hospital of Stockholm by Nordgren [181] established farther evidences for the rheumatic etiology of the disease in the demonstration of an increased seasonal incidence of chorea minor together with that of true rheumatic affections, and in the fact that among the 393 children with chorea, 193 had definite rheumatic histories. Pfaundler,[182] who in his abundant material never encountered chorea and rheumatic affections simultaneously as would be expected according to our pathogenetic conception, speaks of "successive syntropism" of these two diseases.

The necropsy findings by Schroeder [183] of endocarditis and pericarditis with typical Aschoff's nodular cells in the heart muscle of a 20 months old girl who suffered from chorea minor demonstrates again the relationship of the cerebral symptoms and rheumatic infection. The connection between chorea and focal infection became evident also in Yerger's [184] patient, who was promptly cured by adenectomy, the adenoids containing a suppurative sinus (Thornwaldt's disease). Berger [185] noted eosinophilia, averaging 7.6 per cent., with the maximum 26 per cent.

The differential diagnostic significance of Gordon's modified patellary reflex in chorea minor has been tested by Noeggerath [186] in sixty-eight schoolchildren without any indication of chorea, rheumatism or heart lesions. He found in seven children typical, and in several others a less marked, modified Gordon's reflex.

In a series of eighty-eight cases of Sydenham's chorea observed by Hammes [187] definite symptoms of a psychosis existed in about 20 per cent. No uniformity in the mental pictures was noted except that frequently distressing hallucinations were present, conforming to those observed in other toxic psychoses. The mental improvement was followed by rapid and complete recovery. F. R. Fry's remark that chorea is a form of encephalitis or encephalic reaction, the choreic psychoses being a partial manifestation thereof, is supported by the brain sections made by Greenfield and Wolfsohn [188] that presented a pathology very

181. Nordgren, R.: Acta pædiat. **2**:159, 1922.

182. Pfaundler: Ztschr. f. Kinderh. **30**:100, 274, 1921.

183. Schroeder, L. C.: Observations on the Etiology and Pathology of Chorea Minor, J. A. M. A. **79**:181 (July 15) 1922.

184. Yerger, C. F.: Thronwaldt's Disease and Chorea, J. A. M. A. **76**:166 (Jan. 15) 1921.

185. Berger, H. C.: Eosinophilia Occurring in Chorea, Am. J. Dis. Child. **21**:477 (May) 1921.

186. Noeggerath, C.: Monatschr. f. Kinderh. **22**:657 (Feb.) 1922.

187. Hammes, E. M.: Psychoses Associated with Sydenham's Chorea, J. A. M. A. **79**:804 (Sept. 2) 1922.

188. Greenfield, J. G., and Wolfsohn, J. M.: Lancet **2**:603 (Sept. 16) 1922

similar to that in lethargic encephalitis in the early stage; and by the necropsy findings of Gennaro Fiore [189] in two cases, consisting in a disseminated perivascular edematous alveolar encephalitis, most pronounced in the optic thalamus, the corpus striatum and the motor zone of the cortex.

While most authors urge rest and even isolation as one of the prime factors in the management of the disease, Irving [190] attaches great importance to recreation in the country or sea shore, with mild physical activities in the convalescent stage, under trained supervision. Well graded quiet games, etc., resulted in remarkable benefit to 123 choreic children.

Special active movements on a principle similar to that of Frenkel's exercise treatment proved successful in the experience of Krager.[191] Paulian and Dragesco [192] report good therapeutic results from intravenous or intraspinal injections of magnesium sulphate, and Bilotta [193] from intravenous injections of arsphenamin.

TUMORS OF THE BRAIN

Bartlett and Wollstein [194] state that among 4,563 necropsies at the Babies' Hospital, New York, there were nine cases of neoplasms of the brain (0.2 per cent.), the ages of the children ranging from 2 weeks to 3 years. Two neoplasms were located in the cerebrum and five in the cerebellum. All the latter were gliomas of the astrocytoma type. The supratentorial tumors existed in infants; one was a glioma, the other a congenital gliosarcoma. In the infants, the tumors had grown to a considerable size before they gave symptoms, which is explained by the distensibility of the skull and marked adaptability of the young brain to pressure applied gradually.

Convulsive phenomena in tumors of the posterior fossa are rare. Only nine among the forty-five cases studied by MacRobert and Feinier [195] had spasms arising from pressure in the cerebellum. Irregularity and sustained tonicity of the movements are the distinguishing features in comparison with the rhythmic clonic movements of the spasms arising in the forebrain. Forced movements, ticlike spasms in cranial nerves and respiratory embarrassment may develop.

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189. Fiore, Gennaro: Rev. de clin. pediat. **20**:193, 1922.
190. Irving, G. R.: Arch. Pediat. **39**:159 (March) 1922.
191. Krager, R.: Jahrb. f. Kinderh. **95**:261, 1921.
192. Paulian, O. E., and Dragesco, R.: Presse méd. **30**:680 (Aug. 9) 1922.
193. Bilotta, V.: Policlinico (sez. prat.) **28**:545 (April 18) 1921.
194. Bartlett, F. H., and Wollstein, Martha: Arch. Pediat. **39**:386, 1922.
195. MacRobert, R. G., and Feinier, L.: Cerebellar Fits, Arch. Neurol. & Psychiat. **5**:296 (March) 1921.

A paper of paramount interest and based on a wide experience has been written by Dandy [196] on treatment of brain tumors. The author insists that treatment must be based on early and precise localization of the tumor and consist in the complete operative removal. The tumor can be diagnosed and located with accuracy or eliminated by cerebral pneumography. In more than 200 injections, the author never failed to make an accurate localization or to find the tumor at operation.

Menninger [197] testifies to the great value of Dandy's method, but points out certain diagnostic limitations. A number of by-effects of intraspinal insufflation were described by Wideröe.[198] A diagnostically instructive case of the syndrome of the occipitocerebellovertebral angle has been described by Nobécourt and Paraf [199] in a boy who had paralysis of the outer branch of the spinal nerve that innervates the neck musculature, atrophy and subsultus tendinum on the right side, with anesthesia in the area of the right second occipital nerve and slight cerebellar symptoms. Extirpation of the tuberculous tumor at the angle resulted in cure.

Two examples of intracranial dermoid cysts were presented by Horrax [200]; and a case of echinococcus cyst the size of two fists in a boy of 8, filling out the ventricles, by Dévé.[201]

Brain Abscess.—A case of cerebellar diplococcus abscess following mastoiditis, with successful operation for both conditions, is cited by McCoy.[202] The interesting feature was that lethargic encephalitis complicated the clinical picture after the second (cerebellar) operation. Complete recovery ensued.

INFANTILE CEREBRAL PARALYSIS

A unique case of Little's disease in a baby born nonasphyctic by Cesarean section in a case of contracted pelvis has been reported by Tauber.[203] The brain hemorrhage was charged to pressure of the symphysis on the skull during the expulsion period or to overriding of the cranial bones.

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196. Dandy, W. E.: Treatment of Brain Tumors, J. A. M. A. **77**:1853 (Dec. 10) 1921.

197. Menninger, K. A.: Dandy Method of Localizing Brain Tumors by Roentgen Ray, Arch. Neurol. & Psychiat. **5**:438 (April) 1921.

198. Wideröe, S.: Norsk Mag. f. Lægevidensk. **82**:491 (July) 1921.

199. Nobécourt, P., and Paraf, J.: Bull. Soc. de pediat. **20**:327, 1922.

200. Horrax, Gilbert: Dermal Versus Epidermal Cholesteatomas Having Their Attachment in Cerebral Envelops, Arch. Neurol. & Psychiat. **8**:265 (Sept.) 1922.

201. Dévé: Compt. rend. Soc. de biol. **86**:1062, 1922.

202. McCoy, John: Case of Cerebellar Abscess with Very Unusual Features; Operations; Recovery, J. A. M. A. **77**:1333 (Oct. 22) 1921.

203. Tauber: Wien. klin. Wchnschr. **35**:499 (June 1) 1922.

The atonic cerebellar type of diplegia was the subject of a paper by Hempelmann,[204] who reports two cases. In one of them there was distinct asymmetry between the cerebral hemispheres and the two lobes of the cerebellum, due to development defects.

The motor disturbances in infantile encephalopathias of children are classified by Babonneix[205] into various groups. The changes of tonus may be contractures of pyramidal origin, rigidity of pallidal origin and decerebrate rigidity, which possibly originates in the nucleus ruber. The psychomotor disturbances are chiefly paralysis of hemiplegic, paraplegic or localized character. They may be complicated by cerebral syndromes, namely by convulsions, involuntary movements such as athetosis, chorea and synkinesis; by intellectual disorders, hypertonia, awkwardness (paratonia) and also by cerebellar symptoms.

The rarity of hemianopsia in infantile cerebral hemiplegia induced Babonneix and Hallez[206] to report this condition in a child 22 months old.

The end-results of Stoffel operation in seven cases of spastic paralysis, improvement in comfort and appearance of the patient, have been demonstrated before the Philadelphia Pediatric Society by Gill.[207]

Manifestations of infantile pseudobulbar paralysis existed in 2.83 per cent. of 600 applicants for treatment at the University dispensary for speech and voice disturbances at the Berlin Charité. The principal symptoms of the affection, described by Fiebig,[208] were dysarthria, stuttering and jerky pronunciation. The speech was nasal owing to paresis of the soft palate. There was dribbling of saliva, choking and regurgitation of fluids through the nose, with affection of the deglutition muscles. As the focus is in the cortex and not in the oblongata, there is no atrophy or degeneration reaction of the muscles. Other muscular functions which require fine coordination were often disturbed, such as sewing, embroidering, drawing, climbing stairs, etc. Though the patients are frequently mistaken for imbecile, they are by no means intellectually defective. There is a tendency to improvement in the course of years, especially with proper exercise and speech training. A number of interesting examples illustrate the clinical pictures and diagnostic features of the affection.

The complete decerebrate rigidity, observed by Thomson and Piney,[209] in an infant 4 months old had its pathologico-anatomic basis in

204. Hempelmann, T. C.: Arch. Pediat. **39**:411, 1922.
205. Babonneix: J. de méd. de Paris **41**:307, 1922.
206. Babonneix, L., and Hallez, G. L.: Gaz. d. hôp. **95**:997, 1922.
207. Gill, A. B.: Arch. Pediat. **39**:320 (May) 1922.
208. Fiebig, M.: Arch. f. Kinderh. **6**:425 (Feb.) 1921
209. Thomson, A. P., and Piney, A.: Lancet **2**:1105 (Nov. 26) 1921.

extreme softening of the large areas of the cortex, with an almost complete destruction of the deeper layers of the cortex and adjacent white matter and endarteritis obliterans in many vessels.

Wachendorf [210] recalls that the prospects of improvement of paralyses and choreiform and athetoid movements in cerebral infantile paralysis, from operations are unfortunately small. Epilepsy from birth trauma, on the other hand, should be treated early, on the same principle as traumatic epilepsy in general by elimination of scars, cysts, adhesions and removal of bone fragments. The cases of traumatic and jacksonian epilepsy, according to Tilmann, show 18 per cent. of cures. Operations in two of Wachendorf's cases were followed by improvement; in one case, by death in status epilepticus.

BASAL GANGLIONS

Of great interest for the pediatrician is the elaborate and instructive treatise issued by Prof. O. Foerster [211] of Breslau, who, basing his work on a very abundant material, gives an analysis of the physiopathology of striate motor disturbances. He discusses the various forms of Little's disease, dystonia lordotica, myoclonia, certain tics, spastic torticollis and other motor disorders which especially after our great experience derived from epidemic encephalitis have to be connected with the corpus striatum. The destructive pathologic process in Voigt's status marmoratus and possibly in status dysmyelinisatus of the globus pallidus is congenital. A hereditary factor exists in Huntington's chorea, and pseudosclerosis. The etiology of Wilson's disease and of tuberous sclerosis with its occasional striate symptoms is unknown. Other groups of corpus striatum affections are contusions, hemorrhages, as in infantile cerebral paralysis and vascular changes; poisoning with lighting gas, carbon monoxid and possibly manganese; tumors, hydrocephalus and inflammatory processes, as in congenital syphilis and especially in epidemic encephalitis. The author saw generalized athetosis follow epidemic meningitis. Chorea minor is generally admitted to be an infectious brain disease with localizations in the cortex, basal ganglions, nucleus dentatus (Alzheimer) and crura cerebelli. The hypokinetic pallidum syndrome comprises inconstant tremor, increased plastic muscular tone, anomalies of attitude of body and position of extremities, adaptation with fixation rigidity, and catalepsy of limbs. There is a paucity of reactive and expressive movements, slowness, reduced excursion and persistence of voluntary movements and absence of associative movements.

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210. Wachendorf, K.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **34**:64, 1921.
211. Foerster, O.: Ztschr. f. ges. Neurol. u. Psychiat. **73**:1-169, 1921.

The athetotic striatum syndrome, originating in the nucleus caudatus and putamen, comprises athetotic movements at rest, reduction of the plastic muscle tone during the intervals, and anomalies in attitude of trunk and extremities; overextensibility of muscles and a tendency to fixation rigidity. There exist also extensive reactive, expressive and associative movements, which may cause inability to sit and walk.

The components of the choreic syndrome are choreic movements during rest; lessening of the plastic muscle tone; lessening of resistance to stretching and overextensibility of muscles; transitory fixation rigidity, intense reactive and expressive and associative movements during the execution of voluntary movements; inability to sit, stand and walk in severe cases, and replacement of these functions by "massive reactive movements" of the whole body, of choreic character.

One hundred and seventy-three illustrations, including many in children, give a conception of the various motor disorders, demonstrating strikingly extreme distortions of body and limbs in universal and localized forms, compulsory laughing, grimaces, centaur-attitudes of body, torticollis, etc. An elaborate review of the entire subject is given by Crothers,[212] by Bickel [213] and Stertz [214] and others. The first author relates six illustrative observations of his own.

There can be no doubt that among the cerebral disturbances in infants striatal signs may be present ascribable to injury of vessels in the corpus striatum in connection with difficult birth. Babonneix [215] remarks that these lesions may arrest farther development and be followed by persisting conditions.

Although occurring usually in children of the Jewish race, dystonia musculorum progressiva is not confined to that race. Flater [216] reports the disease in a 10 year old gentile girl. In the case of Howe [217] the mother only was of Jewish blood. Another interesting feature in the latter case was that the first disturbances were in the trunk, with shrugging of the shoulders, and a quavering voice, the speech becoming unintelligible within six months. The condition persisted three years and then improved.

Jacob [218] describes typical torsion dystonia in a 14 year old Jewish boy, whose younger brother suffered from symptoms of lenticular

212. Crothers, B.: Lesions of Corpus Striatum in Childhood, Am. J. Dis. Child. **22**:145 (Aug.) 1921.

213. Bickel, G.: Rev. med. de la Suisse Rom. **42**:365, 1922.

214. Stertz, G.: Beihefte z. Monatschr. f. Psychiat. u. Neurol., 1921, pp. 1-96.

215. Babonneix: Ann. de méd. **12**:70 (July) 1922; abstr. J. A. M. A. **79**:1178 (Sept. 30) 1922.

216. Flater, Adolf: Ztschr. f. ges. Neurol. u. Psychiat. **69**:27, 1921.

217. Howe, H.: Neurol. Bull. **3**:253 (July) 1921.

218. Jacob, Charlotte: Arch. f. Psychiat. u. Nervenkrankh. **65**:540, 1922.

character. In a second case, a 12½ year old gentile boy, whose condition was at first diagnosed as torsion dystonia, developed later a clinical picture of pseudosclerosis, with changes in the liver.

Attention should be given to the existence of a myostatic or postural variant of this disease and to the association with fragmentary and almost complete decerebrate rigidity, which has been studied by Wechsler and Brock [219] in six hitherto unreported cases of dystonia musculorum deformans with atypical features.

A study of the types of extrapyramidal rigidity and extrapyramidal pseudobulbar paralysis (akinetic hypertonic bulbar syndrome) in connection with lethargic encephalitis has been contributed by Gerstmann and Schilder [220] of the neurologic clinic at Vienna.

The Magnus and DeKlein phenomenon, a phenomenon of decerebration like decerebrate rigidity, has been analyzed by Meyers [221] in two cases of spastic paralysis, in which the patients presented spontaneous decerebrate attitudes and both types of the cervical reaction. The one child was unable to remain on the back, turning invariably to the left side around onto the abdomen. This condition is referred to generalized atrophy of the cerebellum found postmortem, the left hemisphere being much smaller than the right. The symptoms common to both children are explained by a congenital affection of the lenticular zone, established by Hässin. The author offers a new theory in the physiologic interpretation of the decerebrate attitudes.

Goodhart [222] alone and in company with Tilney [223] studied various postencephalitic deformities of motion by means of film-reels. The motions slowed down served as a basis for what these authors term bradykinetic analysis.

SCLEROSIS

Multiple sclerosis occurs in all ages. It has been described in about a hundred instances in children, occurring even at the early age of 1½, 3 and 4 years. Wechsler [224] adds four cases from his own records, one beginning at 6½, one at 8 and two at 14 years of age. A histopathologic study of acute multiple sclerosis in a girl of 4½ has been published by Neubürger.[225]

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219. Wechsler, J. S., and Brock, S.: Dystonia Musculorum Deformans, Arch. Neurol. & Psychiat. **8**:538 (Nov.) 1922.

220. Gerstmann, J., and Schilder, P.: Ztschr. f. ges. Neurol. u. Psychiat. **70**:35, 1921.

221. Meyers, I. L.: Magnus and DeKlein Phenomena in Brain Lesions of Man, Arch. Neurol. & Psychiat. **8**:383 (Oct.) 1922.

222. Goodhart, S. P.: Postencephalitis Deformities of Motion, Arch. Neurol. & Psychiat. **8**:652 (Dec.) 1922.

223. Goodhart, S. P., and Tilney, F.: Neurol. Bull. **3**:295 (Sept.-Oct.) 1922.

224. Wechsler, I. S.: Statistics of Multiple Sclerosis, Arch. Neurol. & Psychiat. **8**:59 (July) 1922.

225. Neubürger, K.: Ztschr. f. ges. Neurol. u. Psychiat. **76**:384, 1922.

TUBEROUS SCLEROSIS

Freeman,[226] after a review of the literature on tuberous sclerosis, reports his observations in a 6 year old child, giving the necropsy record and the results of the microscopic examination in great detail.

As six cases of this affection presented findings of tumor formations of the papilla optica and retina, Hoeve [227] concludes that such tumors may belong to the syndrome of the disease.

The association of tuberous sclerosis with pubertas praecox is recorded by Krabbe,[228] who holds the view that both conditions are due to one basic factor, a constitutional disturbance in the equilibrium of tissues.

The typical triad of hypernephroma, tuberous sclerosis and adenoma sebaceum in a boy of 10 has been reported by Hyman.[229] The patient had convulsions, marked mental defects and athetosis. Innumerable nodular eruptions on the face and a tumor of the right kidney led to the clinical diagnosis. Nephrectomy revealed hypernephroma.

Diffuse Cerebral Sclerosis (Strümpell's Disease).—The discovery by Giannuli [230] of numerous spirochetes in diffuse brain sclerosis (Strümpell's disease), which were arranged in nests in the cortex, being possibly degeneration forms of *Spirocheta pallida* makes it appear that the affection is a chronic interstitial encephalitis, and that in its pathogenesis not only endogenous but also exogenous elements—*Spirochaeta pallida*—may play a rôle.

SPINAL CORD

An interesting case of permanent disturbance of temperature due to birth trauma of the cervical cord and exemplifying the failure of thermoregulation as in experimental destruction of the cervical cord in animals is cited by Feer.[231] The boy, who was born with a foot presenting, had, in addition to certain signs of a spinal lesion of the cervical region, abnormal temperatures throughout his life with great changes, depending on external conditions, rising, according to circumstances, to or above 41 degrees (Celsius) or dropping to 36 or 35.5. Death ensued on the two hundred and twenty-sixth day. The postmortem examination verified the existence of cord injury from the sixth to the seventh cervical segment.

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226. Freeman, W.: Tuberous Sclerosis, Arch. Neurol. & Psychiat. **8**:614 (Dec.) 1922.
227. Hoeve, J.: Graefes Arch, f. Ophth. **105**:880, 1921.
228. Krabbe, K. H.: Encéphale **17**:281, 437, 496, 1922.
229. Hyman, A.: J. Urol. **8**:317 (Oct.) 1922.
230. Giannuli: Deutsch. Ztschr. f. Nervenh. **71**:306, 1921; Riv. sper. freniat. **44**:287, 1920.
231. Feer, E.: Tag. d. Deutsch. ges. f. Kinderh., 1921; Monatschr. f. Kinderheilk. **22**:203, 1921.

Powers [232] collected seven cases of postdiphtheritic disseminated myelitis or sclerosis and adds one instance of his own, in a boy of 10. The clinical picture was complete but did not terminate in the sclerotic stage, complete recovery ensuing.

Classic syringomyelia, a very rare affection in childhood, is described by De Villaverde [233] in a girl of 15. Parker [234] gives a comprehensive summary of the literature on juvenile tabes and reports seven of his own observations. In all, Westphal's sign was present; in six, diminished sensibility; in four, the pupils were immobile (large); in three cases, Argyll Robertson pupils, optic atrophy, crises, lightning pains, incoordination and hypotonia were present; in two cases, there was incontinence of urine; in one, ataxia. In three patients, the spinal fluid findings, strange to say, were negative. The cases agree well with those of other authors in regard to the paucity of physical signs, the long insidious course and the rarity of ataxia and other features that render juvenile tabes distinct from the adult form.

The view expressed by a number of authors that, in addition to syphilis, as generally recognized, an inherited predisposition, a constitutional inferiority of the nervous system must be assumed as a cooperative factor in juvenile tabes, has found strong support from the studies by Baumgarten [235] of 130 cases gathered from the literature. In 59.38 per cent. of cases with exact anamnesis, metasyphilitic affections, such as tabes or paresis, existed in one or in both parents.

Rosenheck [236] reports typical tabes juvenalis, with pupillary inequality, at the age of 12, as the first sign, subjective disturbances appearing three years later.

An important contribution to neurology was again made by the work of Ayer [237] on cistern puncture and arachnoid block. He worked out a method of combined cistern and lumbar puncture for the determination of subarachnoid block, especially with reference to the early diagnosis of cord tumors.

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232. Powers, H.: Boston M. & S. J. **186**:45 (Jan. 12) 1922.

233. De Villaverde, J. M.: Arch. españ. de pediat. **5**:321 (June) 1921.

234. Parker, H. L.: Juvenile Tabes, Arch. Neurol. & Psychiat. **5**:121 (Feb.) 1921.

235. Baumgarten, C.: Ztschr. f. ges. Neurol. u. Psychiat. **71**:321, 1922.

236. Rosenheck, C.: Juvenile Tabes, J. A. M. A. **76**:572 (Feb. 26) 1921.

237. Ayer, J. B.: Spinal Subarachnoid Block as Determined by Combined Cistern and Lumbar Puncture, with Reference to Early Diagnosis of Cord Tumor, Arch. Neurol. & Psychiat. **7**:38 (Jan.) 1922.

(*To Be Continued*)